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SURGERY

A Monthly Journal Devoted to the Art and Science of Surgery

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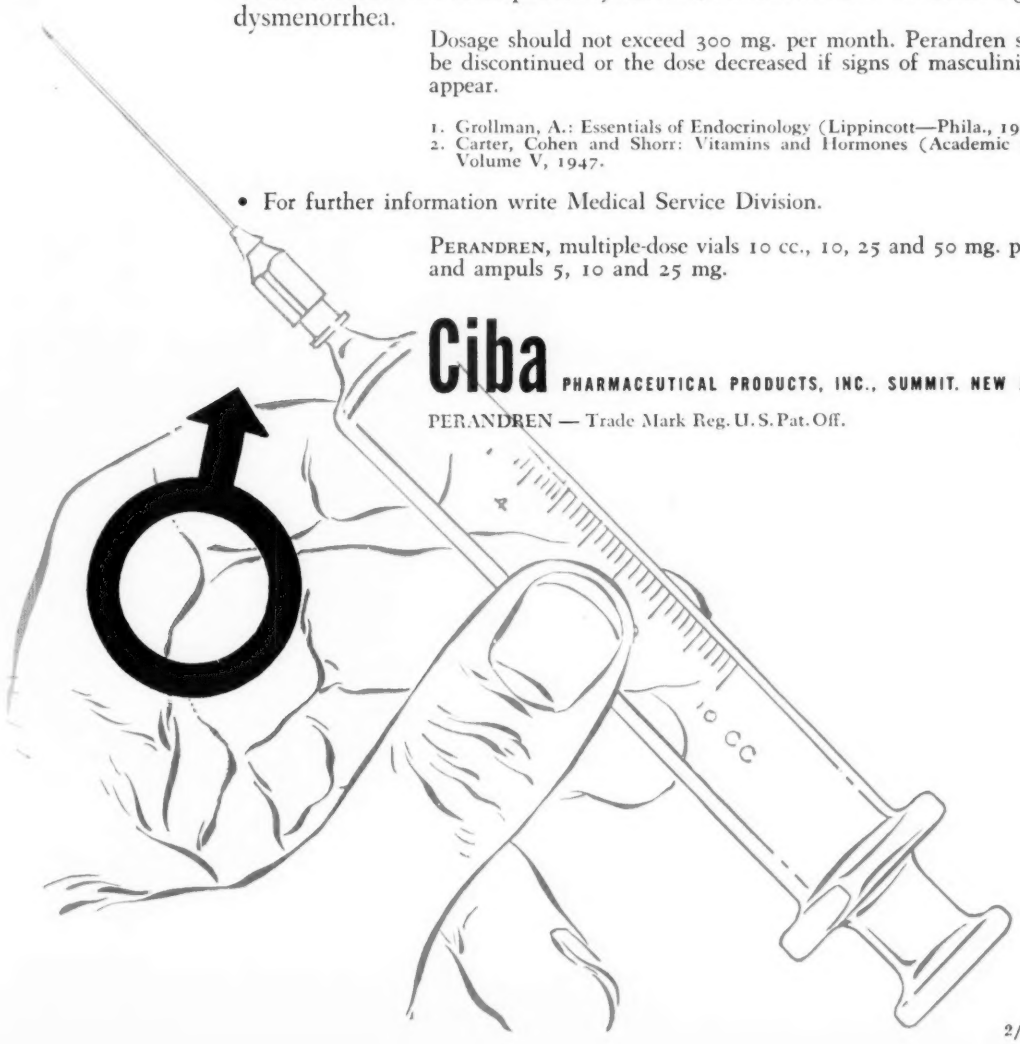

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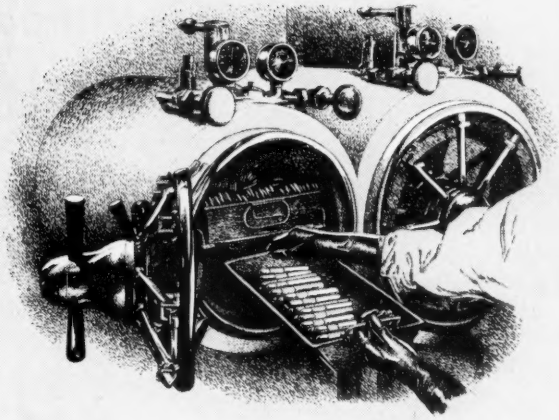

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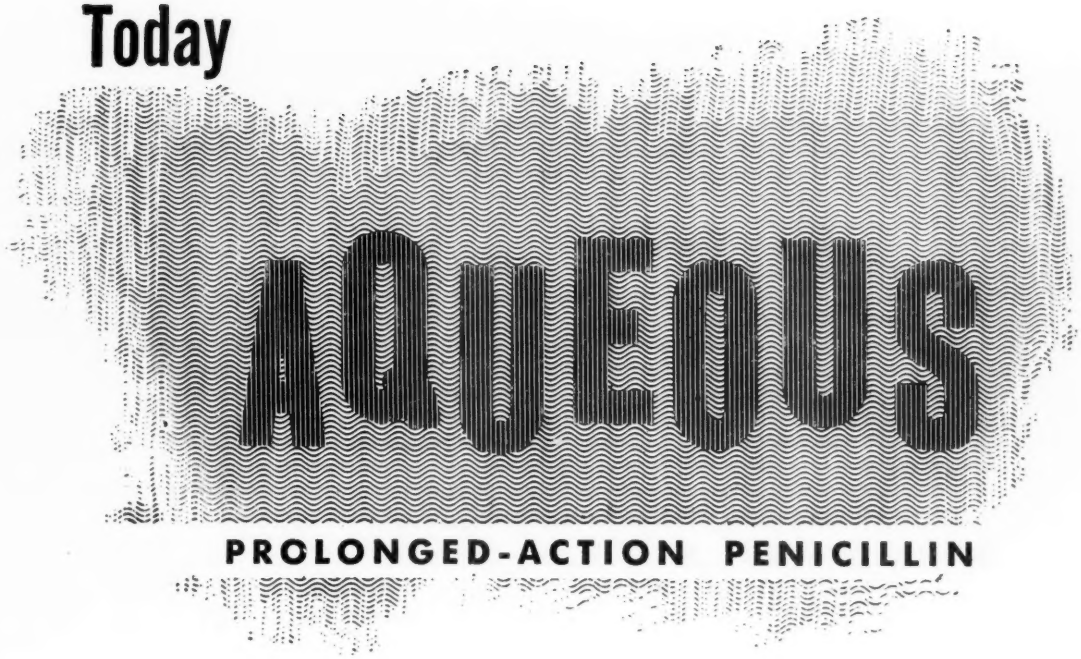

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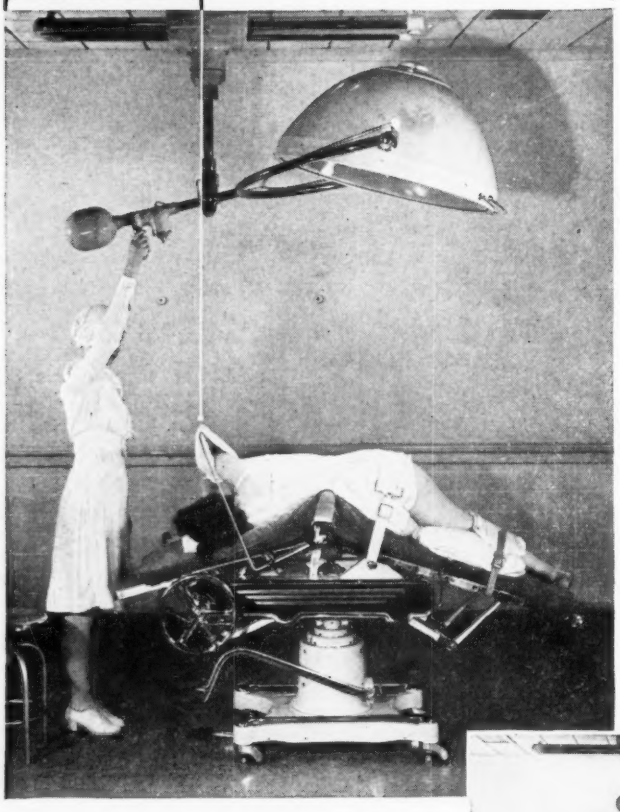

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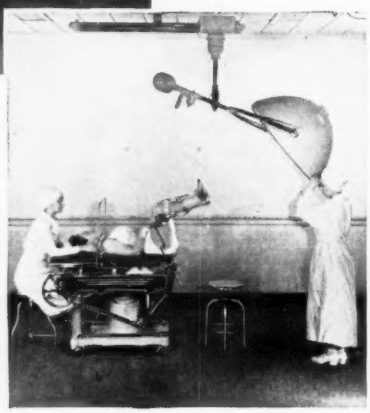

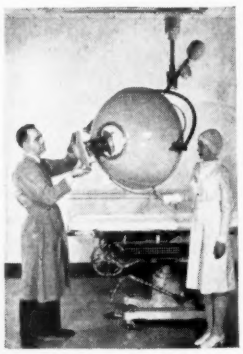

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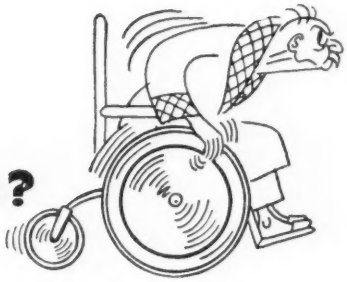

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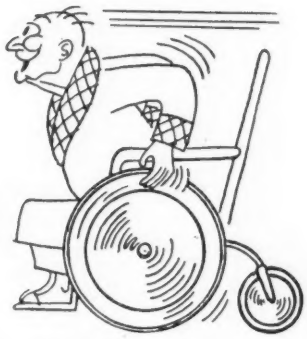

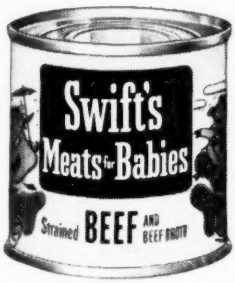

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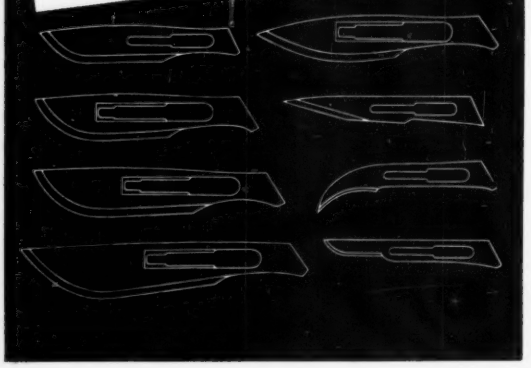

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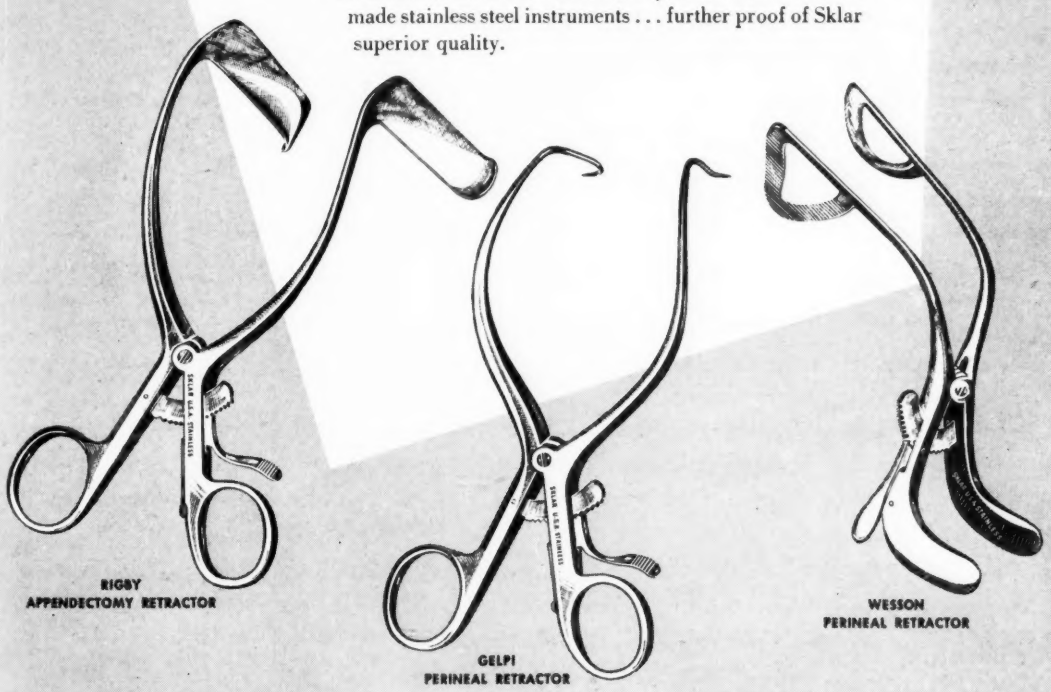

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SURGERY

VOL. 25 MARCH, 1949 NO. 3

Original Communications

THE COLLATERAL CIRCULATION FOLLOWING LIGATION OF THE INFERIOR VENA CAVA

INJECTION STUDIES IN STILLBORN INFANTS

LEWIS S. ROBINSON, M.D., NEW ORLEANS, LA.

(From the Departments of Anatomy and Gynecology, School of Medicine, Tulane University of Louisiana)

THE high mortality associated with suppurative thrombophlebitis of the pelvic veins, the frequent sequelae of infarction and pulmonary embolism, the occasional inefficiency of chemotherapy, antibiotics, and other conservative measures in these conditions, and poor results often obtained by ligation or excision of the ovarian, hypogastric, external iliac, common iliac, or femoral veins alone or in combinations, have resulted in an increased trend toward a broader type of operative procedure which has decidedly reduced the mortality and morbidity associated with this condition, namely, ligation of the ovarian vessels and the inferior vena cava. Ligation of the vena cava alone has also been effective in the therapy of phlebothrombosis and/or thrombophlebitis of the iliac vessels.

The currently proved operative management of suppurative pelvic thrombophlebitis has stimulated an interest in the collateral circulation following ligation of the inferior vena cava below the renal veins.

Purpura[1a] concluded, on the basis of experiments on dogs, that sudden ligation of the inferior vena cava in man was incompatible with life unless this vessel had been stenosed or effaced in part prior to ligation in order to permit gradual development of the collateral circulation. However, clinical observations have disproved this belief. It is now known that life is compatible with sudden complete ligation in the lower one-third of the inferior vena cava or gradual obliteration of the vessel.[2-46] Numerous clinical[2, 17, 29, 34, 40-43, 46-59] and experimental[1b, 23, 40, 57, 60-65] observations have shown that the inferior vena cava can be suddenly obliterated at any point below the renal veins, usually without

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provoking noticeable circulatory trouble, due to the ability of the collateral circulation to return adequately the venous blood.

Haller,[4] in 1768, reported a case of obliteration of the lower one-third of the inferior vena cava, and since then others[5-19] have reported similar cases and some have described the collateral circulation. Morgagni,[20] in 1769, reported a case of obliteration of the inferior vena cava and its tributaries and a number of others[3, 21-33, 37] have described obliteration of other portions of the inferior vena cava. Recovery has been reported in the majority of cases[2, 35-36, 38-46, 66-68] in which the inferior vena cava had been ligated after its injury in the course of nephrectomy or removal of an abdominal tumor. In 1903, Houzel[36] concluded that ligation of the inferior vena cava was without great danger, and was indicated, if the vessel were wounded or had to be resected to remove a tumor. Pfaff,[42] in 1926, concluded that its ligation below the renal veins was safer than suture of the wounded vessel. Ligation of the inferior vena cava has become increasingly popular since about 1906 in the surgical treatment of suppurative thrombophlebitis of the pelvic veins and phlebothrombosis and thrombophlebitis involving the common iliac veins.[17, 34, 47-55, 58-59, 69-78]

Battle, (79), in 1887, stated that the signs and symptoms which follow blocking of the inferior vena cava are dependent upon whether this is acute or gradual in onset, whether a thrombus forms in the main vein or extends into the inferior vena cava from other veins, and whether the thrombus extends upward or downward. According to many authors,[5, 10, 12-19, 28-33, 37, 56, 70, 79-85] the most marked symptom is edema of the legs, followed by formation of a plexus of enlarged and varicose veins over the abdomen, flanks, lumbar region, and lower extremities. The direction of the blood flow in these large varicose veins is from below upward. Occasionally varicocele, hemorrhoids, and dilated veins in the penis and scrotum are present. Pain is inconstant and usually abdominal or lumbar. Occasionally there is ulceration of the lower extremities, some engorgement of the liver, and rarely albuminuria.[37, 56] It is not uncommon to find that the edema of the lower extremities abates as the supplementary circulation is established.[37] However, these signs and symptoms are usually seen in cases of thrombosis or compression of the vena cava by tumors. Obstruction of the inferior vena cava, even when sudden or complete, is not necessarily accompanied by either edema or superficial collateral circulation.[24, 56, 86] This is especially true following surgical ligation of the vessel.[2, 17, 29, 34, 37-43, 46-59] In most of the cases only a transitory edema has been present and development of the superficial collateral circulation has been a rare occurrence.

Brackertz[57] concluded from clinical and experimental studies that if the inferior vena cava is ligated suddenly just below the renal veins, the collateral circulation is formed mainly by the lumbar veins and their communications. If the ligation is made just above the iliac veins, the superficial veins dilate to form the collateral circulation. He stated that this is confirmed by the observation that the abdominal veins are not enlarged when it is necessary to ligate the inferior vena cava following its injury during a nephrectomy. Wakefield and Mayo[17] concluded that if there is no intra-abdominal pressure, infection,

etc., the deep collateral channels are adequate in the development of a fully compensated collateral circulation when the inferior vena cava is obstructed below the renal veins. However, if the deep channels do not develop to the extent necessary to pass the blood to the upper part of the vena cava, a fully compensated circulation cannot be developed by addition of the superficial channels.

The absence of signs of serious vascular upset in cases of complete and sudden obstruction is probably due to the multiplicity of small collateral channels, all of which are capable of prompt even if slight dilation, thus establishing rapid compensation. The period of adjustment is indicated by the transient edema.[37]

The chances of postoperative complications after ligation of the inferior vena cava are minimized by postoperative lumbar sympathetic blocks or by interruption of the sympathetic lumbar chain at the time of operation.[52, 55, 71-73, 87]

Collins[52, 55] and his co-workers found in their series that in some patients edema of the feet and legs developed immediately after ligation of the inferior vena cava, but subsided after a period of time. They reported their follow-up examinations to have revealed no edema of the extremities, no dilated peripheral veins, no evidence of varicosities of the vulva or vagina, and no hemorrhoids.

Physiologic studies made on patients at Tulane University by Ray and Burch[88] following ligation of the inferior vena cava revealed:

1. Edema of the legs and feet varied from none to marked immediately after operation. In all instances in which edema occurred it usually disappeared by the end of the sixth week. In only two patients did it persist for as long as eight months.
2. The pressure in the veins of the dorsum of the foot remained elevated in all cases. There was a decline over a period of four to five years, but in only two cases did it fall to the upper limits of normal. The pressure in the abdominal veins was increased.
3. There were no varicosities, or nail or skin changes. The abdominal venous plexus was unusually prominent and the direction of blood flow was cephalad in all cases.
4. The volume of pulse deflections as measured by a plethysmograph was found to be reduced.
5. The rate of water loss from the skin of the tip of the right second toe and pretibial area was normal.
6. There were no demonstrable ill effects from ligation of the inferior vena cava.

According to Burch[89] it is not surprising that only a few patients develop mild chronic edema following ligation of the inferior vena cava, as it is practically impossible to produce edema in the legs of dogs by ligation of the inferior vena cava.

EXPERIMENTAL STUDIES

Human Cadavers.—There has been but little experimental work using human cadavers in the study of the collateral circulation of the inferior vena

cava. In an effort to study the collateral circulation during the autopsy of a case with obliteration of the inferior vena cava from the renal veins to the common iliac vessels, Sappey and Dumontpallier[5] in 1862, injected the venous system with tallow via the femoral and axillary veins. They divided the collateral circulation into anterior, lateral, and posterior channels, and presented an excellent description of the veins concerned and their communications. In injection studies on cadavers, Cholwoz[90] showed that at a pressure of 150 mm. Hg the collateral veins will become sufficiently functional in 90 per cent of the cases, especially in women who have borne children. Recently Batson's[60, 91] work on the function of the vertebral veins and their role in metastasis has emphasized the potentialities and the collateral communications of this system in the cephalad flow of blood. He performed a very complete study of the vertebral veins in 100 cadavers by injecting a radiopaque material into the dorsal vein of the penis. More recently Northway and Greenway[92] have studied the collateral circulation following ligation of the inferior vena cava at a point in the lower one-third of the vessel. In their experiments they used unprepared cadavers and injected the venous system via the internal saphenous vein with a thin suspension of red lead. Following roentgenograms they dissected the venous channels concerned in the collateral circulation. The results of these experiments are presented under the discussion of the collateral circulation.

Animals.—Considerable time and effort has been spent throughout the years in experimental work on animals in an attempt to study the collateral circulation following ligation of the inferior vena cava at different levels.

In 1669, Lower[93] published the first account of experimental ligation of the inferior vena cava in an animal. Brunton,[94] in his paper on the pathology of dropsy, cited Ranvier's[95] experiments of ligating the vena cava of the dog and the observation that it did not always produce edema of the legs. Purpura,[1b] in 1898-1899, on the basis of experiments on the dog, concluded:

1. That survival is possible after the ligation is made in one stage at a point between the common iliac and the hepatic veins. Life is more difficult to maintain if the ligation is made above the renal veins.
2. Stenosis of the vein prepares a terrain favorable to absolute obliteration.
3. If obliteration is effected immediately below the renal veins, compensation is effected by the external anterior vertebral veins, the left ovarian-uterine veins, ureteral veins, veins of the abdominal wall, and inferior mesenteric vein. If the obliteration is above the renal veins, the circulation re-establishes itself by the vessels clinging to the veins of the parenchyme and capsule of the kidney, by the external anterior vertebral veins, veins of the abdominal wall, and inferior mesenteric vein.
4. It is the same for man. Stenosis of the inferior vena cava favors obliteration of the vessel.

Raynaud[23] found less albumin in the urine of rabbits and dogs after ligation of the inferior vena cava below the renal veins than after ligation above them.

Gosset and Lecene,[62] in 1904, experimenting on the dog, concluded, in part:

1. The inferior caval system of the dog is exactly comparable to that of man.
2. Ligation of the trunk of the inferior vena cava below the renal veins is not accompanied by any pathologic phenomenon, not even a trace of edema of the legs.
3. Ligation of the trunk of the inferior vena cava above the renal veins provokes grave lesions of the kidneys. In spite of the existence of anastomoses between the renal veins and the caval and azygos systems, death always results from this ligation.

Offergeld[61] published experiments on ligation of the inferior vena cava between the renal and the common iliac veins on four rabbits, five cats, and two dogs. All the cats died, but the other animals survived without edema or severe disturbance in sensitivity or temperature. Clinically he noted slight extension of cardiac dullness to the left, and strengthening of the second sound at the aortic valve. The only other effect of the operation was a transitory disturbance of the motor nerves of the legs (tremor).

Leotta,[63] following his experimental studies on animals in 1907-1908, reported:

1. Ligation of the inferior vena cava above the renal veins is always followed by death resulting from renal lesions.
2. Ligation at the level of attachment of the two renal veins in such a manner that one renal vein remained above and the other below the ligation was fatal in 33 per cent of the animals and well tolerated in 67 per cent. Circulation was re-established by the two azygos veins, the ascending lumbar veins, the reno-azygos trunk, and the lumbar vein of Legras, and by the epigastric veins and the internal mammary veins.
3. Ligation of the inferior vena cava made below the renal veins, no matter at what point, was well supported.

Bejan and Cohn,[40] in 1911, did similar experiments on the dog. They concluded:

1. Ligation of the inferior vena cava below the attachment of the renal veins is compatible with survival and good health.
2. Ligation at the level of the attachment of the renal veins and including one of the veins in the ligature is well tolerated. The circulation for the involved kidney re-established itself easily by the veins at the superior pole, as well as by anastomosis with the corresponding suprarenal veins, the abdominal parietal veins, and the diaphragmatic veins.
3. Ligations made immediately above the renal veins and the left suprarenal vein are equally compatible with life. They are well tolerated and are compatible with good health. Circulation re-establishes itself by the plexus of veins at the superior poles of the two kidneys

and from there by the suprarenal veins directly to the inferior vena cava at the right; and at the left when the suprarenal vein has been included in the ligation by a large anastomosis of this vein with the diaphragmatic veins, the abdominal parietal veins, the lumbar veins, and more distantly with the azygos and the vertebral veins. They pointed out that Purpura[1b] was the only one who had demonstrated survival in a similar case of abrupt ligation of the inferior vena cava above the renal veins.

4. Ligations made higher than the right suprarenal vein always resulted in death.

Brackertz[57] observed from animal experiments that the collateral circulation is formed mainly by the lumbar veins, when the inferior vena cava is suddenly ligated just below the renal veins; if the ligation is made just above the junction of the common iliac veins the superficial veins of the abdomen form the main collateral circulation.

In 1929 Polkey[64] studied the phenolsulfonphthalein output after ligation of the vena cava below the renal veins in dogs, and found the kidneys to be normal and to function well. His findings were in accord with both experimental and clinical investigations showing that the kidneys do not suffer, except temporarily perhaps, when the inferior vena cava is ligated below the level of the renal veins. Edema or slight albuminuria may develop, but neither is common, and both are usually transitory.

Batson's[60] injection experiments on the monkey have done much toward demonstrating the extensiveness of the potential collateral circulatory system and the importance of the vertebral veins following ligation of the inferior vena cava. In his experiments he injected the dorsal vein of the penis with a radiopaque mass and observed that the material passed into the pelvis, followed the pelvic wall, and ascended the trunk by means of the inferior vena cava. He felt that this was the ordinary course of flow during trunk muscle inactivity. When a towel was tied about the abdomen to simulate physiologic conditions of increased intra-abdominal pressure and the injection was repeated, while some of the material entered the inferior vena cava, it ascended only part way; some of the solution entered the vertebral system of veins through which it passed the zone of compression and then flowed into the vessels of the thoracic spine and out into the lower intercostal veins. These experiments show the venous flow to be from the pelvic veins into the vertebral vein system during abdominal straining or compression.

Whittenberger and Huggins,[65] in 1940, concluded from their experiments that ligation of the inferior vena cava above both renal veins caused death in dogs in a few hours from surgical shock, due to accumulation of blood from the kidneys in the posterior portion of the body. When oblique ligation of the inferior vena cava between the kidneys is done, allowing development of collateral veins in one kidney, subsequent complete high ligation of the vena cava is well borne; it is not followed by shock or interference with renal function in the kidney with adequate venous drainage.

Most authors agree that the mortality is high and the kidneys are seriously affected after ligation of the inferior vena cava above the renal veins. In the event of recovery, however, the kidneys resume good functional activity.

ANATOMY

Seib[96] dissected the azygos system of veins in 200 cadavers and studied the origin of the azygos and hemiazygos veins. The azygos usually arises from the dorsal aspect of the inferior vena cava at the level of the second lumbar vertebra. The hemiazygos usually arises from the dorsal asject of the left renal veins, sinistral to the aorta and opposite the upper part of the second lumbar vertebra. Both the azygos and the hemiazygos then continue cranially to enter the thorax, passing through the crura of the diaphragm. As they reach the body of the twelfth thoracic vertebra they are joined, laterally, by the common trunk of the subcostal and ascending lumbar veins, medially, often by a persistent right or left subcentral vein. Up to this point the azygos or hemiazygos may be appropriately named lumbar azygos or lumbar hemiazygos, but with these reinforcements their diameter usually becomes considerably augmented and they may be designated azygos or hemiazygos, as they continue cranialward in the thorax. One is not always able to define the lumbar portion of the azygos or hemiazygos veins. Therefore, Seib considered the azygos and hemiazygos as being formed from several roots, one of which would be the lumbar azygos or lumbar hemiazygos veins. He defined the origin of the azygos and hemiazygos veins, in terms of adult descriptive anatomy, as follows: The azygos and hemiazygos veins arise in the thorax from one, two, or three roots; a lateral, the common trunk of the ascending lumbar and subcostal veins; an intermediate, the lumbar portion of the azygos or hemiazygos veins; and a medial, the persistent right or left subcentral vein.

Keen,[97] in reporting a case of thrombosis of the inferior vena cava, was of the opinion that if the inferior vena cava becomes obstructed, the right side of the body will be more embarrassed than the left, because on the right side the embryonic veins have become incorporated in the inferior vena cava and are not available if the vessel becomes thrombosed. On the left side, remnants of the embryonic pattern may be present as small anastomotic vessels which can become dilated in case of necessity. In his case this offered a reasonable explanation of the difference in compensation on the two sides as evidenced by varicosities of the right leg. Latimer and Virden[98] reported a case of complete absence of the inferior vena cava, and described the collateral circulation and its embryologic interpretation.

As may well be expected, the veins concerned in the collateral circulation following ligation of the inferior vena cava are numerous. Haller,[4] in 1768, was probably the first to describe the collateral channels in a case where the inferior vena cava was obliterated between the iliac and the renal veins. In his case the circulation was re-established by the uterine veins, the right utero-ovarian veins, and the right ureteric vein, the latter being the principal vein in the collateral circulation. Baillie,[21] in 1793, described the collateral circulation by the lumbar and azygos veins, but the thrombosis involved the upper two-thirds of

the inferior vena cava. Cruveilhier[22] showed that the principal means of establishing circulation between the inferior vena cava and the superior vena cava are the azygos and vertebral system of veins, and the superficial veins of the trunk. Cruveilhier stated that the great azygos vein which is generally regarded as the chief communication between the two vena cavas is not indispensable because of the presence of the anterior and posterior vertebral plexus of veins. He had observed cases in which either the superior or the inferior vena cava had been obliterated without noticeable increase of the azygos vein, or even without edema of the extremities. Wilson,[99] in 1804, described the vertebral, portal, and azygos routes in detail in a case of thrombosis of the lower two-thirds of the inferior vena cava. Bidault,[9] in 1804, and Castelnau,[6] in 1841, described the superficial veins making up the collateral circulation in cases of obliteration of the lower one-third of the inferior vena cava. Hodgson[7] also described the superficial veins in a similar case, but also included dilation of the lumbar veins and the epigastric veins.

Hallet[8] described a case of obliteration of the lower one-third of the inferior vena cava in a woman, and stated that the veins of the uterus were dilated and formed a plexus to aid it, of which the veins of one side communicated with veins of the opposite side. He found the collateral circulation to be established by the uterine veins, the left utero-ovarian vein, the left ureteric vein, and the left renal vein. The left utero-ovarian vein was quite dilated, but the parietal veins were not. This case is similar to that reported by Haller.[4]

Sappey and Dumontpallier,[5] in 1862, reviewed the literature and described the collateral circulation in obliteration in different types of obstruction of the inferior vena cava. In their reported case of obliteration of the inferior vena cava in its lower one-third, the collateral circulation was described in detail following injection and dissection of the veins at autopsy. Their description included the superficial and deep veins of the anterior and lateral abdominal walls and thorax, and four venous currents on the posterior part of the trunk which included the lumbar veins, the azygos system, the portal system via the inferior mesenteric vein, and the vertebral system of veins. They mentioned equal dilation of all the deep and superficial veins of the posterior wall of the abdomen, and "especially the extra- and intra-vertebral veins which bring back the blood in the superior one-half of the ascending vena cava and in the azygos veins." Peacock,[24] Turpin,[19] Thomas,[100] Stillman and Carey,[12] Skillern,[14, 84] and Keen[97] have also mentioned the importance of the vertebral plexus of veins in the formation of the collateral circulation in occlusion of the inferior vena cava. Nicaise[101] and Poirier[3] pointed out that if a ligation were made at a point on the trunk of the inferior vena cava and the iliac veins were injected, the fluid easily arrived in the heart and the same result could be obtained by ligating the inferior vena cava in several places at once; that the amplitude of the anastomotic paths which supply this venous trunk is great enough that the injected liquid would reach the heart even if the trunk had been obliterated in its totality; and that the venous anastomosis would also connect the two ends of the inferior vena cava.

Poirier[3] stated that the collateral circulation following ligation or obliteration of the inferior vena cava was extensive, and included the azygos veins, lumbar and sacral veins, diaphragmatic veins, vertebral veins, renal veins, veins of the anterior abdominal wall, and the portal vein.

Other authors have also described the collateral circulation in varying degrees of detail in reporting cases of obstruction or thrombosis of the inferior vena cava.[10, 12-19, 28, 30, 79-86]

More recently Sparks and Fox[31] and Keen[97] have described the collateral circulation in two cases of complete obstruction of the inferior vena cava from its beginning to the hepatic veins. Thomas,[100] in 1893, showed in table form the veins involved in the establishment of the collateral circulation following various types of obstruction of the inferior vena cava. These tables were modified in Pleasants'[56] classical work on the subject in 1911.

Batson's[60, 91] experiments on animals and the human cadaver demonstrated a set of valveless, lexiform, longitudinal, venous channels that join the cranial venous sinuses to the pelvic veins without the intermediation of the lungs. Batson considered the vertebral veins as a system of valveless vessels which carry the blood under low pressures, consisting of the epidural veins, the perivertebral veins, the veins of the thoracoabdominal wall, the veins of the head and neck, and the veins of the walls of the blood vessels of the extremities. He further considered this system to be a storage lake because the vascular bed is much larger around the vertebral column than required by the part in which it is found. He pointed out that the vertebral venous system is constantly subject to arrests and reversals in the direction of flow of blood and that it parallels, connects with, and provides by-passes for the portal, the pulmonary, and the caval system of veins.

In 1944 Northway and Greenway[92] studied the vena cava and its collateral circulation, using unprepared cadavers. The inferior vena cava was doubly ligated above the bifurcation and radiopaque material was injected into the system via the saphenous vein. They found the external and internal vertebral plexi to form a vast network of intercommunicating valveless veins within and without the spinal canal. These form an adequate communication between different levels of the inferior caval systems themselves as well as between the inferior and superior caval systems. They concluded that this extensive system is more than adequate in its collateral potentialities, and emphasized that ligation of the inferior vena cava merely serves to place a closed valve within the vena cava, that portion of the vein proximal to the ligature being readily filled from the communicating branches of the vertebral plexus.

PERSONAL OBSERVATIONS

Eight stillborn infants were used in this injection study of the collateral circulation following ligation of a point on the lower one-third of the inferior vena cava. These specimens had been held in a morgue refrigerator more than forty-eight hours, and all had some degree of post-mortem clotting. The weights varied from about one pound to thirteen and one-half pounds. The technique used was similar to that of Lieb[102] with some of the modifications of Duff and

More.[103] The injection apparatus employed was essentially that described by Narat, Loef, and Narat[104] in their article on the preparation of multicolored corrosion specimens. However, in most of the cases a small electric motor-driven pump which produced a pressure of from 100 to 120 mm. Hg was used. In two cases a large syringe with a cord plunger was substituted. The syringe was satisfactory for injection of Neoprene* for the plunger did not stick as does a glass or metal plunger. However, a constant pressure is difficult to maintain by such a method and one is likely to rupture the small mesenteric or retroperitoneal veins.

It was difficult to find a suitable vessel or combination of vessels to perfuse through in an effort to drive the blood from the venous system. As a rule, edema of the tissues would occlude the circulation after two to three hours. After several vessels and combination of vessels were tried, it was found that the best results were obtained by perfusion through a femoral or axillary vein, allowing the water to drain from the umbilical artery or the femoral or axillary vein of the opposite side. When ascites or edema closed these routes of drainage, the water was allowed to escape through an incision made in the right atrium. The axillary veins were ligated and the right atrial incision was repaired prior to injection.

It was found that unless the azygos vein was ligated at its termination and the superior vena cava ligated at the heart, a retrograde flow of the injection mass filled the veins of the thorax and neck. Naturally this filling did not give a true picture of the collateral circulation. It was further determined that the ascending aorta should be ligated to prevent filling of the arterial system.

Considerable difficulty was experienced in finding a suitable vein for cannulation, perfusion, and injection. Due to the small size and the thin wall of the veins of most of the specimens, small cannulas were unsatisfactory because they tore the vein wall or had so small a tip that very little pressure could be used in perfusion or injection. Larger cannulas were difficult to place in the small femoral or saphenous veins. A No. 10 rubber T tube was placed in the inferior vena cava in several specimens and was fairly satisfactory. Good results were obtained by perfusing through the tube and injecting through it following ligation of the inferior vena cava below the renal veins. However, on small specimens it was difficult to place such a tube in position. The best results were obtained by the use of 15 to 18 gauge needles with rounded points. These needles could be inserted in the femoral or internal saphenous veins without difficulty, and tied and left in place. By this method a minimum of manipulation in the abdomen was necessary. It was found early in the experiment that the handling of the intestine and the solid viscera, or probing about to isolate and cannulate the inferior vena cava, caused considerable leakage of the injection mass from the liver and mesenteric and retroperitonal veins, which made dissection difficult and obscured the outline of the veins by x-ray.

In Case 1, synthetic liquid latex (yellow) was used for the injection mass. Results were fairly satisfactory except that the mass was not radiopaque, was

*The Anode Co., Akron, Ohio.

too thick to fill the smaller veins well, and was quite friable after fixation, so that the veins broke easily during dissection. In the rest of the cases blue radiopaque Neoprene was used. The mass was very thin, filled even the smallest

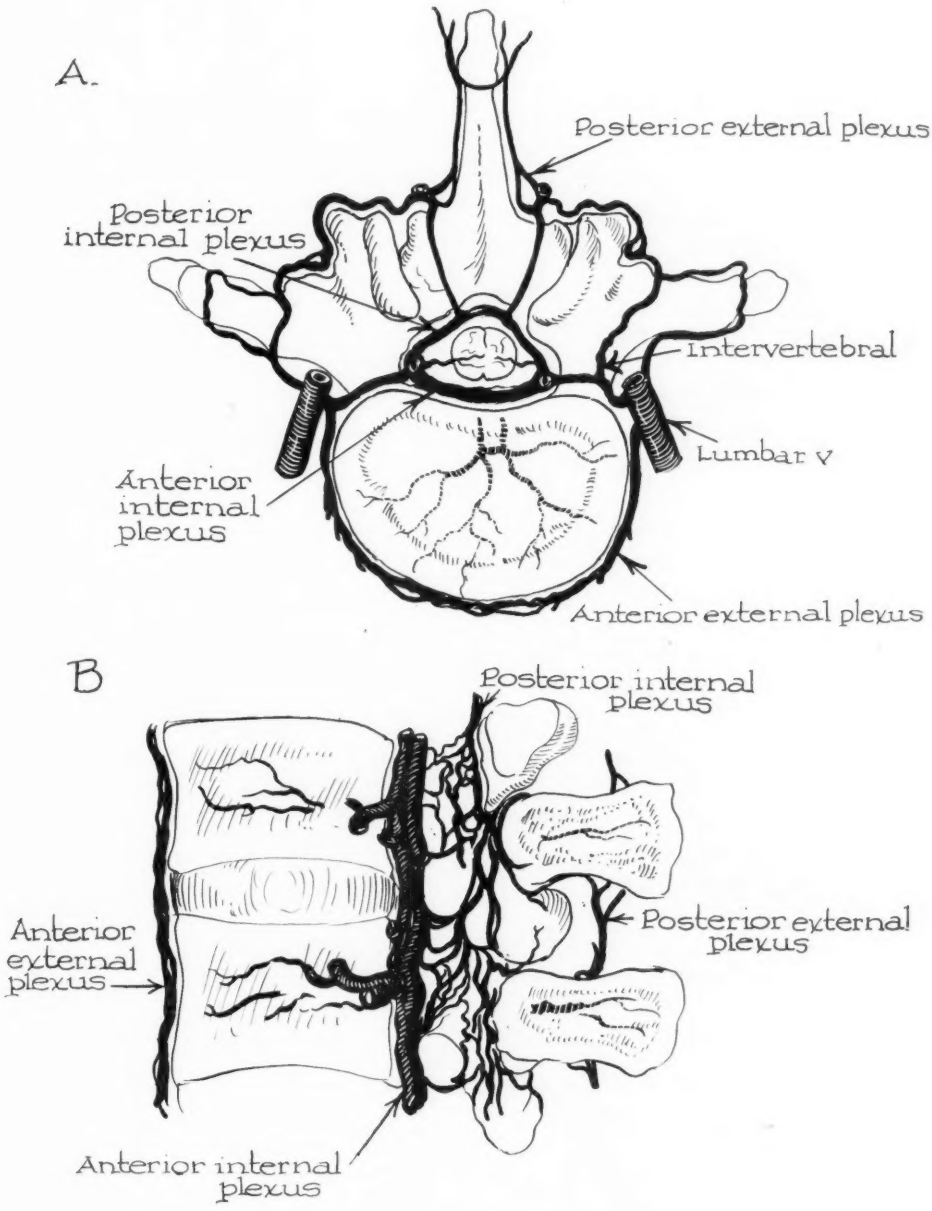

Fig. 1.—Veins of the vertebral column.

veins, and was quite elastic following fixation, making isolation and identification of veins easy if reasonable care was used in the dissection. However, the radiopaque material settles out rapidly if allowed to stand, and one must agitate the bottle containing the solution constantly during injection. Even so, the mass

does not give clear outlines of the injected veins on roentgenograms. The addition of 100 c.c. of skiodan to approximately 600 c.c. of Neoprene in an effort to make the solution more radiopaque in Case 8 made no noticeable difference in opacity.

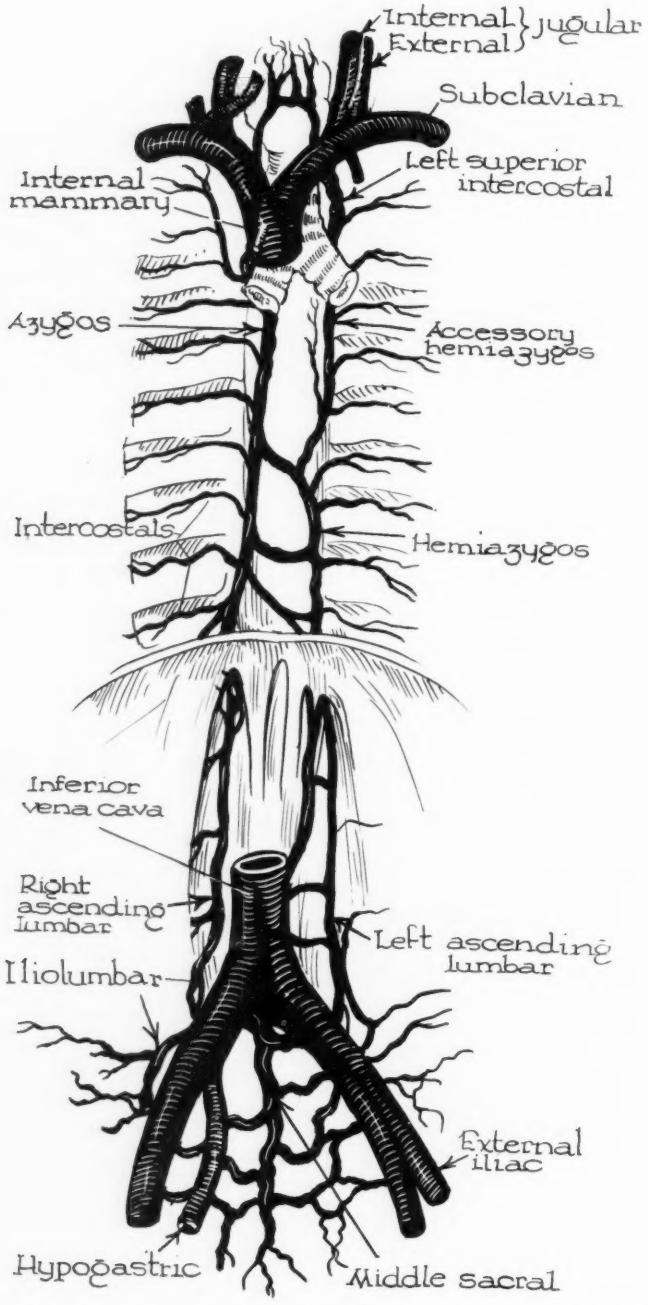

Fig. 2.—Veins of the walls of the thorax, abdomen, and pelvis.

Leakage of the injection mass is easily controlled by touching the vessel or organ with cotton pledgets saturated with glacial acetic acid. It was found that a solution of 10 per cent acetic acid and 10 per cent formaldehyde was more satisfactory for fixing the Neoprene in the stillborn infant, and better preserved the specimens for roentgenograms and dissection. Following fixation

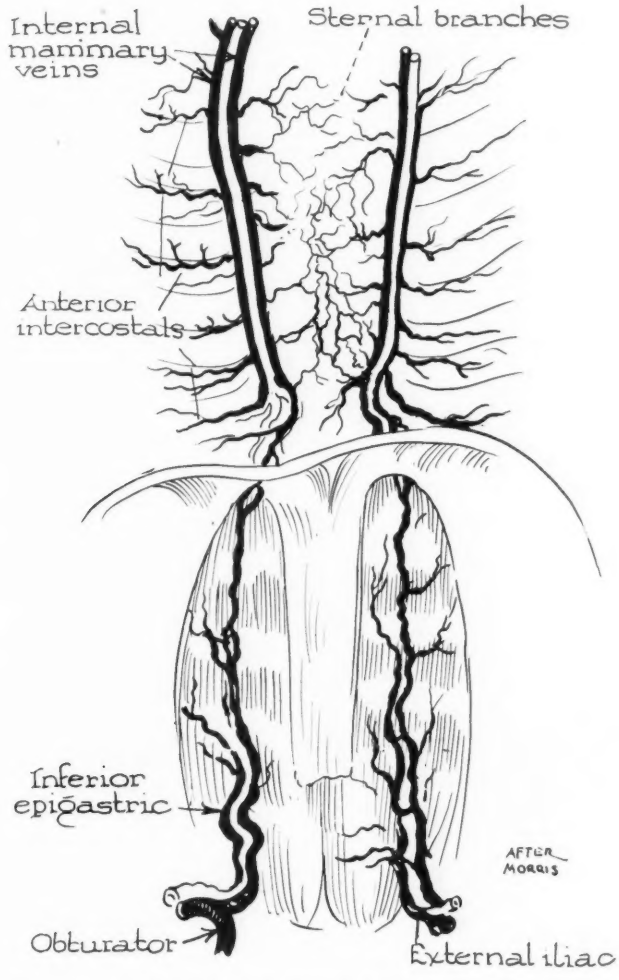

Fig. 3.—Veins of the walls of thorax and abdomen, posterior view.

of the injection mass the liver, lungs, and small and large intestines were carefully removed to give clearer roentgenograms of the veins of the thorax and abdomen. The inferior mesenteric vein and hemorrhoidal veins were preserved in all specimens. Pins were placed in the larger veins before the x-ray views were taken, in an effort to establish landmarks to aid in identification of the collaterals.

Corrosion specimens were obtained in Cases 2, 4, and 5. The technique used was similar to that of Lieb[102] except that the specimen was placed in a tightly covered vessel containing concentrated U.S.P. hydrochloric acid and put

in an incubator at 52° for a period of forty-eight hours. Corrosion specimens were not too satisfactory due to incomplete filling of the vessels, thrombi in the veins which caused organs to be lost and hence destroy landmarks, and the fact that the casts of the innumerable fine veins stuck together and formed an inseparable mass or occluded the larger venous casts by their presence. It was noted in all cases that the vertebral plexus was made up of a multitude of fine, intercommunicating, longitudinal veins from the sacrum to the cranial vault. They had a multitude of communications throughout their course. The three corrosion specimens added little to the experiment except to show that there does exist a very adequate collateral circulation between the proximal and distal portions of the inferior vena cava when it is ligated between the renal veins

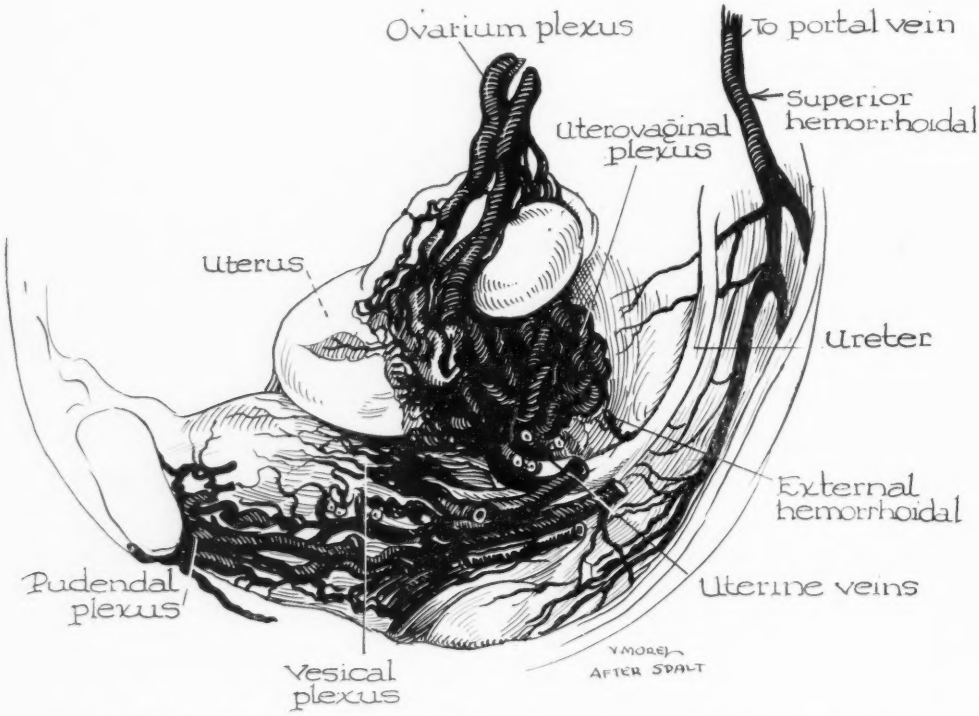

Fig. 4.—Veins of the viscera of the pelvis and anastomoses with the portal system.

and the junction of the common iliac veins. In this series, x-ray studies of the injected vessels were unsatisfactory due to the inadequacy of the opaque material in the injection mass, the poor filling in some of the cases, and the small size of the vessels concerned. It was noted that as far as x-ray, dissection, and corrosion studies were concerned, the small three- to six-pound specimens gave the best results. The smaller specimens are more difficult to prepare for injection, but due to their small size and the absence of excessive adipose tissue, they are easier to dissect and more satisfactory roentgenograms are obtained.

For convenience of description and identification, an attempt has been made to divide the veins into seven groups corresponding, in so far as possible,

to a combination of the usual system of classification[105-107] and a regional separation. These are, namely, (1) the vertebral plexus of veins; (2) veins of the thoracic wall; (3) veins of the neck; (4) veins of the walls of the abdomen and pelvis; (5) veins of the viscera of the abdomen and pelvis, including the portal

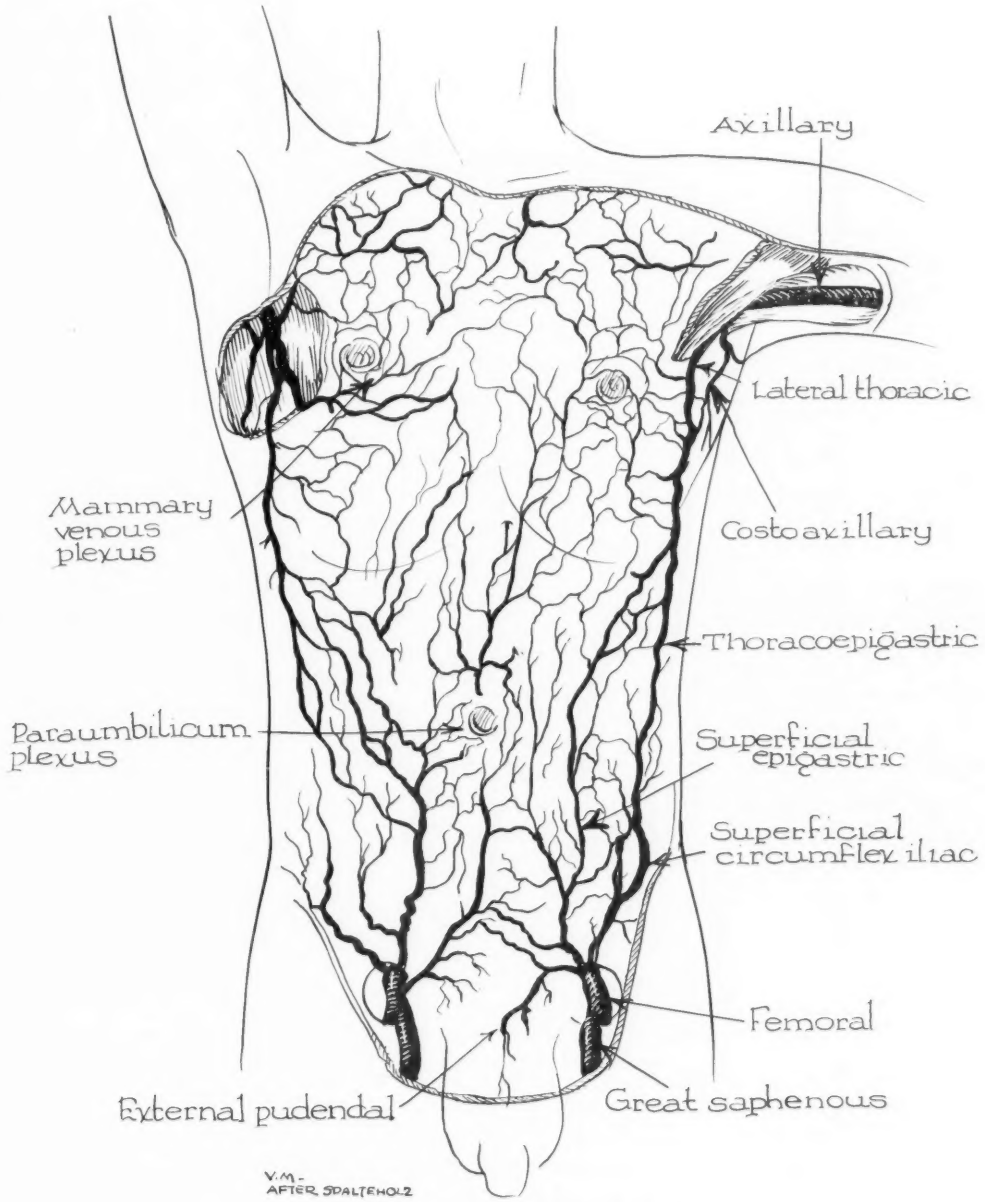

Fig. 5.—Superficial veins of the trunk.

system; (6) veins of the thoracic viscera; (7) veins of the subcutaneous tissue of the abdomen and pelvis. It is to be noted that all seven groups of veins intercommunicate either directly or indirectly, and that all are concerned with the establishment of the collateral circulation following ligation of the inferior vena

cava. Figs. 1 to 5 illustrate the main veins concerned in the collateral circulation. As a result of the injection studies, the vertebral, azygos, and portal systems of veins are considered the most important routes of the collateral circulation following ligation of the inferior vena cava. The superficial veins of the trunk are the least important of the collaterals.

Observations during the injection of Neoprene into the saphenous or femoral veins showed the vertebral and azygos systems to fill immediately. Also, the visceral veins of the abdomen and pelvis filled readily as evidenced by the injection of the mesenteric veins, the liver, and organs in the pelvis. Studies following injection showed the vertebral plexus of veins, the veins of the neck, the veins of the thoracic wall (including the azygos system), the veins of the walls of the abdomen and pelvis, and the veins of the abdominal and thoracic viscera to be well filled, and in most cases to communicate. However, the superficial collaterals were found to be only partially filled, if at all, and anastomotic connections, as evidenced by the injection mass, were infrequent. These results coincide with clinical and experimental evidence concerning surgical ligation of the inferior vena cava, that is, (1) the vertebral, azygos, and portal systems of veins play a large role in the collateral circulation following ligation of the inferior vena cava; the superficial veins are rarely dilated; (2) following ligation of the inferior vena cava at any point in the lower one-third, the proximal and distal ends are still filled and still communicate due to its vast collateral system; (3) after ligation of the inferior one-third of the inferior vena cava the collateral channels are more than adequate to return effectively the blood from the lower portions of the body to the heart without noticeable changes in the circulation.

SUMMARY

1. The history of obstruction and ligation of the inferior vena cava is reviewed.

2. The signs and symptoms found in obliteration by disease of part or all of the inferior vena cava, and following ligation in the lower one-third of the vessel, are briefly discussed.

3. The experimental work on human cadavers and animals in the study of the collateral circulation of the lower one-third of the inferior vena cava is reviewed. The conclusions regarding the results of the various experiments are enumerated.

4. The collateral channels concerned and their description by various workers, following obliteration or ligation of various portions of the inferior vena cava are presented, along with a review of the literature on this subject.

5. The procedure of injection studies in stillborn infants is reported. Dissection, corrosion, and roentgenogram studies were made of specimens.

6. The collateral circulation following ligation of a point on the lower one-third of the inferior vena cava is presented.

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TETRAETHYLAMMONIUM CHLORIDE IN EVALUATION OF LOWER EXTREMITY ARTERIAL DISORDERS

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AS INDICATIONS for lumbar sympathectomy have broadened, the need for accurate preoperative evaluation of patients with lower extremity arterial disorders has been increasingly apparent. Oscillometry has been little used where accurate opinions are needed; plethysmographic studies appear most useful where institutional study is possible. Skin temperature studies are probably more valuable than oscillometry and less tedious than plethysmography. Lumbar sympathetic block with procaine is a tedious, painful procedure. Spinal anesthesia allows simultaneous study of both lower extremities but does not permit evaluation of exercise tolerance.

Apparently, response to temporary autonomic blockage with tetraethylammonium chloride† answers the need for a clinically practical method of accurate study; it does not require hospitalization and is easy of interpretation. Lyons, Moe, Neligh, Hoobler, Campbell, Berry, and Rennick[1] concluded that action of the tetraethylammonium ion is best explained as a blockade of the sympathetic and parasympathetic ganglia. A further study[3] summarized results following 1,500 injections of the drug. Cooper, Robertson, and Dennis[4] recently concluded that chemical blockade of autonomic ganglia by the tetraethylammonium ion may be of value in acute arterial deficiency following arterial injury as a substitute for other methods of sympathetic interruption, or as a preliminary to sympathetic interruption. They believed that favorable results incident to use of tetraethylammonium chloride are due to removal of collateral vessel vasoconstriction and resulting vasodilatation.

During the study reported in this paper, tetraethylammonium chloride was used as the sole agent to produce autonomic blockade. Two hundred intravenous injections of the drug were given in doses ranging from 400 to 500 mg. Intramuscular administration of the drug accounted for an additional 100 injections. Only one alarming blood pressure depression occurred, this in an 80-year-old man with auricular fibrillation, congestive failure, and popliteal artery thrombosis. We now consider tetraethylammonium chloride safe for clinical use but do continue to follow, in general, the precautionary measures and routine practiced by the original investigators. One patient received 34,000 mg. intramuscularly over a period of ten days with no unfavorable reaction. One bed patient required an indwelling catheter for eight days during prolonged usage of the drug.

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Twenty-six patients were subjected to 28 lumbar sympathectomies during this study. The operation, in each case, was done for functional or occlusive lower extremity vascular disease. The transverse incision employed for sympathectomy is similar to that used for removal of the gall bladder, although the inner end is one or more inches lower and the approach is retroperitoneal. The fourth lumbar ganglion was spared in the first 20 operations but has been resected since that time. When possible, the first ganglion has been removed in addition to the second, third, and fourth. No mortality has occurred. One reactive wound infection required resection of the scar with removal of silk sutures. We now consider it inadvisable to use nonabsorbable sutures in patients with thromboangiitis obliterans.

TECHNICAL METHOD

Skin temperature tests were carried out with the patient recumbent and unclothed in a draftless room, the temperature varying from 74 to 84° F. High room temperatures perhaps gave falsely high basal skin temperatures, but we do not believe results are significantly modified in differential responses. Each patient was left uncovered for 30 minutes prior to the tests and usually felt slightly cool at the time determinations were made. Corrections were made for each variation in room temperature occurring between skin temperature readings.

Initial thermocouple recordings were made from symmetrical locations on the forearms, body, and lower extremities, including the first and fifth toes. Tetraethylammonium chloride was then injected intravenously over a period of 60 seconds. Blood pressure and pulse rate were recorded each 5 minutes and skin temperature readings were again made 30 minutes following autonomic blockade. Maximum increase in temperature usually occurred between 25 and 30 minutes after the intravenous injection. The patient was then allowed to dress and his "exercise tolerance" over a measured distance was noted. On assuming the upright position from recumbency, the patient invariably was momentarily dizzy but within 5 minutes could usually walk rapidly without help. More than one-half our patients were those walking on crutches or with impending gangrene. In these, it was necessary to rely on the patients' opinions of subjective change after injection of the drug. Comparative venous filling times were done when feasible. Subsequent intramuscular injections of the drug were often used when it seemed desirable to obtain a better opinion of subjective change following chemical blockade; in some instances the patient was not told the nature of the drug being injected. When intramuscular administration was employed for diagnostic purposes, 500 mg. of tetraethylammonium chloride were injected in one buttock every 4 hours. Occasionally, 1000 mg. were injected every 4 hours, half in each buttock.

Patients experiencing good subjective responses invariably stated that the leg and foot seemed much warmer, less tense, and had "more life." They could walk faster and claudication appeared later. It must be admitted that vasodilatation following one intravenous injection rarely lasts objectively more than 40 minutes. Subjectively, the improvement rarely persisted over 2 hours. Results in alleviating true "ischemic rest pain" were very poor. Skin temperatures

following tetraethylammonium chloride intravenously averaged 2° C. lower than those usually obtained following surgical ganglionectomy. We have, however, had an occasional case in which one poor objective response to chemical blockade was followed by later satisfactory responses.

ANALYSIS OF OPERATIONS

Group I. Functional Lower Extremity Vascular Disease (8 cases, 9 lumbar sympathectomies).—Seven patients were under 40 years of age. All had cool, moist feet, nocturnal burning pain, and the symptoms had followed trauma. The postoperative results were classified as excellent in 6 and satisfactory in 2 cases. The minimum period of follow-up was 7 months. In 8 patients the skin temperature response following autonomic blockade was excellent. In 7 patients the subjective response was excellent; in 1 patient it was equivocal. In no case did the symptomatic relief following tetraethylammonium chloride exceed 18 hours.

Group II. Thrombo-angiitis Obliterans (12 cases, 12 lumbar sympathectomies).—The 12 patients were less than 51 years of age and the diagnosis was carefully established in each case. Five patients had previously been subjected to thigh amputation. Following sympathectomy the results were considered excellent in 3, satisfactory in 4, and failures in 5 cases. The minimum period of follow-up was 5 months. The failures were followed by leg amputation in 3 and by thigh amputation in 2 cases. In the 3 patients with results classified as excellent, the preoperative skin temperature response to autonomic blockade and the subjective response were equally good. In the 4 patients with satisfactory results after operation there was little preoperative temperature improvement on autonomic blockade, but in the 4 cases all had obtained good subjective response to tetraethylammonium chloride. In the 5 failures no preoperative subjective improvement had occurred, yet 3 of these patients had fair preoperative temperature responses.

Group III. Arteriosclerotic Lower Extremity Arterial Disease (6 cases, 7 lumbar sympathectomies).—The 6 patients were more than 50 but less than 65 years old. Following lumbar sympathectomy the results were considered satisfactory in 3 cases and poor in 3 cases. The minimum period of follow-up was 6 months. In the 3 patients with satisfactory results the preoperative skin temperature response following tetraethylammonium chloride agreed with the subjective improvement. In the 3 cases with poor operative results, good temperature response had occurred prior to operation in 2 cases but the subjective response to autonomic blockade had been absent.

COMMENT

It is generally agreed that none of the commonly used tests for the presence or absence of vasomotor constriction is completely satisfactory. Clinical evaluation of the average patient has required hospitalization and laborious study with methods which are not exact. The subjective response to any method of examination should be superior if it confirms the impression of the examiner, who is able by experience to evaluate objective information obtained by inspection and palpation. In our hands, tetraethylammonium chloride has given an average

skin temperature response only 2° C. lower than that obtained by lumbar sympathectomy. The immediate subjective response of increased "exercise tolerance" following chemical autonomic blockade has been striking in our experience. For the patient who cannot be ambulated, repeated intramuscular therapy has relieved the presenting symptoms when marked vasomotor constriction was present. It is possible that the incorporation of tetraethylammonium chloride in a medium permitting much slower absorption and excretion would answer the desirability of maintained uniform response for a number of hours following a single intramuscular injection. We believe that this drug can be used in the physician's office and that it will give him an opportunity to evaluate many types of peripheral vascular disease with much greater ease than has heretofore been possible.

SUMMARY AND CONCLUSIONS

1. Tetraethylammonium chloride has been used as the sole agent to produce autonomic blockade in the preoperative study of a group of patients with lower extremity peripheral vascular disorders. The drug has been found safe to use and no serious reaction has occurred in a series of 300 injections.

2. In the preoperative evaluation of a series of 26 patients later subjected to lumbar sympathectomy, skin temperature studies have been compared with "exercise tolerance" or other subjective response in the same patients. Subjective change following chemical autonomic blockade has usually predicted the expected result following lumbar sympathectomy better than have the skin temperature studies.

3. We now depend on the subjective response to chemical autonomic blockade in determining the advisability of lumbar sympathectomy.

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THE SURGICAL THERAPY OF DUODENAL ULCER

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DURING the past thirty years, the surgical therapy of duodenal ulcer has undergone many radical changes. Up to 1920, the surgical method of choice was gastroenterostomy. At that time, Haberer first suggested the procedure of partial gastrectomy *with removal of the ulcer*-bearing portion of the duodenum as a definitive curative remedy. Following this more radical operation, there was noted a marked reduction in the percentage of gastrojejunal ulcer, a complication which followed the operation of simple gastroenterostomy with distressing frequency. To Lewisohn and Berg belongs the credit for introducing Haberer's operation to America and for insisting, in spite of almost universal opposition in the early years, that its proper performance was based on sound physiologic principles and would result in a high percentage of permanent cures.

Since Dragstedt's physiologic studies on the effects of vagotomy and his recommendation that vagotomy be used as a definitive remedial measure for duodenal ulcer, there has developed great confusion in the profession at large as to what constitutes the best advice to a patient suffering from duodenal ulcer of the type requiring surgical treatment. Many practitioners have stated to us that, instead of simplifying the problem, the introduction of vagotomy as a method of therapy has had the effect of confusing the entire problem of the treatment of this disease. It is not uncommon today to have patients ask questions of this sort: "Am I a candidate for this new operation of nerve cutting? 2. What are the relative merits of gastrectomy and vagotomy from the standpoint of immediate mortality and long-term symptomatic relief? 3. Has this new operation been done long enough to warrant definite opinions about the permanent cure of ulcer? and finally, 4. Doctor, do you advise me to have this new operation of vagotomy instead of gastric resection?"

In an attempt to clarify for ourselves some of the issues involved, we undertook the analysis of 187 cases of duodenal ulcer, all patients subjected to a partial gastrectomy, in the private practice of the senior author. We should state that approximately 60 per cent of these constituted semiprivate material, which, since the introduction of the various Blue Cross Plans of Group Hospital service, approximates closely the type of patient seen on the wards. We believe that the patients in this series constitute an excellent cross section of the general population of a large active urban hospital, and therefore, do not represent, as one might suppose, a small select group of people. The period of observation of these patients was thirteen years, 1934 to 1947, and all follow-up observations were on a personal examination basis by one or the other or both

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of us. Many have had at least one late postoperative gastric analysis to determine the presence or absence of free HCl.

There is no need to enlarge at great length on the indications for surgery in the therapy of duodenal ulcer. We believe, as do most others, that ulcer is primarily a medical disease and that surgery becomes necessary in the presence of (1) intractability to thorough adequate medical treatment, (2) repeated hemorrhages, (3) perforation into the peritoneal cavity or penetration with the production of an exoduodenal pocket, and (4) obstruction due to scarring or inflammation. We are firmly convinced from our experience that the operation of gastroenterostomy alone has no place in the therapy of this disease with one exception and that is a benign obstruction accompanied by a low acidity in an elderly patient where the risk of a gastric resection would be somewhat increased. Curiously enough we have not had a single such instance in this series.

Our concept of the rational surgical therapy of duodenal ulcer has been and still is, in spite of the recent emphasis on vagotomy, based on the well-known physiologic studies of gastric digestion in normal and ulcer patients. In the order of importance, there are three factors which influence the progress of duodenal ulcer: (1) hyperacidity, (2) hypersecretion, and (3) hypermotility. From experimental observations, it is well known that there are two major factors in the production of gastric acidity and secretion: (1) the hormonal, the elaboration of a hormonal substance, in the mucosal glands located in the pyloric antrum and pyloric ring which is absorbed and stimulates the acid-forming cells in the fundus and upper part of the stomach to secrete hydrochloric acid, and (2) the cephalic, a psychic or neurogenic stimulus propagated from the midbrain through both vagus nerves for direct action on the gastric secreting cells and the musculature of the organ.

In our opinion, the operation of partial gastrectomy, properly performed, will produce an absence of free HCl in a high percentage of the patients and will not be accompanied by the undesirable motor difficulties which frequently follow the operation of vagotomy. It is important to emphasize what we mean by a properly performed gastrectomy. It is unimportant how high the stomach is transected provided the line of division is above the re-entrant angle, thus making for removal of approximately two-thirds of the organ. What is most important, and we believe this to be the crux of the entire operation, is that the surgeon must be sure that he has removed the entire pyloric antrum *and pyloric ring*. In the vast majority of instances this means that the ulcer-bearing portion of the duodenum *must* be removed. The demonstration of the entire pylorus and ulcer in the removed specimen indicates that a complete operation has been performed. One of us (J. H. G.) has emphasized for many years that, more often than not, it is impossible to delineate the pylorus in vivo as a separate structure because of scarring and/or periduodenal and peripyloric inflammation with marked distortion of the local anatomy resulting therefrom. If, therefore, one is to make sure that he has removed the entire pyloric ring, he must, in the vast majority of cases, remove the ulcer-bearing part of the duodenum. This insistence on removing the ulcer area often makes for increased technical

difficulty, but we are firm in the opinion that, with careful meticulous dissection, the thorough operation can be consummated in almost 100 per cent of the cases with a commendably low mortality. In our series, there was only one instance where it was felt that insurmountable technical difficulties precluded the execution of the complete operation. An exclusion operation of the Finsterer type was done. The patient developed a gastrojejunocolic fistula six years later. A subsequent three-stage Pfeiffer operation which included pylorectomy resulted in complete relief with the production of an anacidity. This patient was operated upon some years ago. We are of the opinion that if the operation were done today, a thorough resection would be effected. If the surgeon is positive that he can remove the pyloric ring and if the ulcer is located at the bend of the first portion of the duodenum or in the second portion, then there is no harm in leaving the ulcer behind, provided that a good closure of the duodenal stump can be effected. We do not go so far as to insist that second portion ulcers must be excised with the attendant technical maneuvers directed to the common duct which is frequently inextricably involved in such cases. We believe the operation, under such circumstances, is made unnecessarily complicated. Gastrectomy with excision of the pylorus will result in spontaneous healing of the excluded ulcer. There were three instances in this series of second portion ulcer treated in this way.

One cannot overemphasize the importance of a general agreement among surgeons as to what constitutes a properly performed gastrectomy. The recent literature is replete with instances of misinformation resulting from the lumping together of cases in which the patients have had the radical operation and others who have been accorded the incomplete Finsterer exclusion procedure. In the latter group, many observers state that the antral mucosa has been excised, thereby assuming that the radical operation has been consummated. Anyone who has had any considerable experience with the operative treatment of this disease knows that, in the complicated cases with penetration and extensive periduodenal and peripyloric inflammation and distortion, it is impossible to be sure that this coring out process thoroughly removes the mucosa from the pyloric ring. To us, this has been a serious defect in this procedure and we have therefore never employed it. To be sure of complete removal of the pyloric antrum and ring frequently requires a painstaking and meticulous, often difficult, dissection. This part of the operation, we wish to emphasize, is the essential feature of a thorough gastrectomy.

It is unnecessary at this time to describe in detail the various steps of the operation as we perform it. Every surgeon, as his experience increases, develops for himself a set technique with the purpose of (1) simplifing the various steps of the operation, (2) minimizing postoperative complications, (3) decreasing operative mortality, and (4) increasing the percentage of permanent symptomatic relief. In most instances we perform a gastrectomy of the Hofmeister type with a retrocolic gastrojejunal anastomosis. If the gastric stoma, after transection, is small, a Polya anastomosis is carried out. We are careful, when confronted with a dilated stomach, the result of pyloric obstruction, to take into

account the subsequent contraction of the stomach when fashioning the size of the stoma. For the duodenal stump we use as a first layer a continuous Connell suture of fine silk which is reinforced with one or two additional layers of interrupted stitches of the same material. We believe that since the advent of fine nonabsorbable suture material in this situation, there has been a marked reduction in the incidence of postoperative duodenal leakage. For the gastrojejunal anastomosis, we use fine chromicized catgut in two layers. Whenever there has been extensive retroperitoneal dissection in the region of the duodenum or pancreas, we always insert one cigarette drain to the subhepatic region. This is removed on the seventh or eighth day. We are firmly convinced that the use of a drain where extensive denudation has resulted has materially decreased our incidence of postoperative abdominal complications. There can be no question of the fact that some localized pancreatic necrosis follows extensive dissections made necessary by posterior penetrating ulcers. When catgut is used for the duodenal closure, the liberation of pancreatic enzymes causes rapid digestion of the catgut resulting in a high incidence of duodenal stump breakdowns. Fine nonabsorbable suture material is excellent insurance against this complication. Drainage of the subhepatic area is an added safeguard.

We prefer posterior gastrojejunal anastomoses because it is our feeling that fewer postoperative mechanical difficulties result. The posterior anastomosis appears to us to be a more physiologic one from the standpoint of more efficient emptying of the stomach and the avoidance of a long proximal jejunal loop. The care of the duodenal stump is of prime importance in this operation. It is our belief that close attention to a few of the basic principles of surgery will result in fewer duodenal stump breakdowns. By this is meant (1) avoidance of tight sutures, (2) reduction of the number of sutures to a minimum, (3) avoidance of large bites in the duodenal wall, and (4) complete hemostasis. Not infrequently when a posterior wall ulcer has perforated into the pancreatic head, the surgeon is confronted with a paucity of duodenal wall tissue posteriorly. Further dissection distally might result in injury to the retroduodenal portion of the common duct or the papilla of Vater region. Under such circumstances, we have, for many years, utilized the pancreatic bed of the ulcer as the posterior part of the suture line, approximating the anterior duodenal wall to it and then reinforcing this initial layer with additional sutures bringing the duodenum to pancreatic capsule. This has proved to be a valuable maneuver in thirty-two cases.

We have personally had little experience with the operation of vagotomy as a definitive procedure for duodenal ulcer. It has been our good fortune to follow the work of Colp and his associates on the gastric service of this hospital. They have given and are giving the operation in its original sense and subsequent modifications a complete and thorough trial. Their findings have been of considerable interest and appear in their published papers. It is our distinct feeling from our observations that the results of vagotomy, with or without gastroenterostomy and with a negative insulin test meal indicating complete vagal section, do not justify the abandonment of the operation of gastric resection. We believe that as time goes on and these patients are followed over a

sufficiently long period of time, there will be noted a rising incidence of either recurrence of the duodenal ulcer or gastrojejunal ulcer where gastroenterostomy has been added at the time of vagotomy. We believe this to be so because the operation of vagotomy alone does not eliminate the hormonal factor of gastric secretion, and as time progresses the later may become the dominant cause of the reappearance of hyperacidity and reactivation of the original ulcer. It appears more logical to utilize *both* procedures, namely, gastrectomy *and* vagotomy under certain circumstances, as in cases of marked hyperacidity or those with repeated hemorrhages. As experience increases, the simultaneous combination of both operations may prove to be the ideal one. For the present (since October, 1947) we are employing infradiaphragmatic vagotomy as an adjuvant procedure to gastrectomy in all patients where the preoperative acidity has been unduly high and in all cases where hemorrhage has been the dominant preoperative symptom. It is our feeling at this time that if gastrectomy, properly performed, can be carried out with a reasonably low mortality, a negligible postoperative morbidity, and an excellent rate of long-term cures (with especial reference to the late development of gastrojejunal ulcer), then it should be considered the primary operation of choice, and that vagotomy should be employed as an adjuvant procedure only. However, we would like to emphasize the fact that, in our limited experience with gastrojejunal ulcer following adequate gastrectomy, complete vagotomy alone seems to be a specific curative remedy. This has been the experience of most workers in this field of surgery.

The analysis of the cases in this series has amply confirmed the views expressed in the foregoing paragraphs.

ANALYSIS OF CASES

The total series consisted of 187 patients. There were 154 males and 33 females, a ratio of almost 5 to 1. An analysis of the age grouping reveals the interesting fact that the majority were in the fifth and sixth decades (Table I). It is again an interesting fact that the majority of the patients in this series had ulcer sympoms for five to twenty years (Table II). Our case analysis

TABLE I. AGE INCIDENCE OF PATIENTS IN THIS SERIES

YEARS	NUMBER OF PATIENTS
20 to 30	16
31 to 40	48
41 to 50	70
51 to 60	45
61 to 70	7
Over 70	1

TABLE II. DURATION OF SYMPTOMS BEFORE OPERATION

YEARS	NUMBER OF CASES
1 to 5	40
5 to 10	44
10 to 15	52
15 to 20	31
20 to 25	14
Over 25	6

indicated that the vast majority of these patients had had many medical "cures" and that surgical relief was sought when intractability to conservative treatment supervened. In 123 cases, the main presenting symptom was ulcer pain. The indication for surgery in the remaining 64 patients was the history of repeated hemorrhages. Of these, 26 never experienced pain and 38 had the usual ulcer pain between bleeding episodes (Table III).

TABLE III. PREDOMINANT SYMPTOMS AT TIME OF FIRST EXAMINATION

SYMPTOMS	NUMBER
Pain	123
Repeated hemorrhages without pain	26
Repeated hemorrhages with pain	38

We have included in these statistics all secondary cases in which the patients were operated upon elsewhere for duodenal ulcer or its sequelae.

In 16 instances, there had been a previous free perforation with simple suture of the ruptured ulcer. Significant obstruction at or near the site of the ulcer was noted in 23 patients. Marked penetration into the pancreas or gastrohepatic ligament with pronounced anatomic distortion was disclosed in 35 cases. Multiple or kissing ulcers were demonstrated 12 times in this series. We have included in this group of 187 patients 11 instances of gastrojejunal ulcer and 3 of gastrojejunocolic fistula, on the basis of a gastroenterostomy previously performed elsewhere (Table IV). In the first, operation consisted of gastrectomy after disconnection of the old gastroenteric anastomosis, resection of the involved jejunum with end-to-end anastomosis, and a retrocolic gastrojejunostomy. In the second group, a three-stage Pfeiffer operation was carried out. There was no mortality in this group of 14 patients (Table IV).

TABLE IV. OTHER PERTINENT DATA

DATA	NUMBER OF PATIENTS
Previous free perforation	16
Significant obstruction	23
Penetration into pancreas or liver	35
Gastrojejunal ulcer on basis of previous gastroenterostomy	11
Gastrojejunocolic fistula on basis of previous gastroenterostomy	3
Gastrojejunocolic fistula following incomplete gastrectomy in our series	1
Previous pyloroplasty	3

With very few exceptions, the anesthesia of choice in this series has been gas-oxygen-ether with frequent tracheal intubation. With an expertly administered general anesthesia, often helped by curare, we believe that thorough relaxation can be obtained. We are of the opinion that there is far less risk from all standpoints with general than with spinal anesthesia. The incidence of disturbing pulmonary complications in this series had been remarkably low.

The postoperative abdominal complications are listed in Table V. There were 4 instances of duodenal leak, and, in 1, it caused the death of the patient. In 2, the leak was temporary and spontaneous closure occurred. In the fourth patient, it was necessary after six months to explore the stump of the duodenum

TABLE V. POSTOPERATIVE ABDOMINAL COMPLICATIONS

COMPLICATION	NUMBER	DURATION	INFLUENCE RECOVERY
Severe gastric atony	3	10 days	No
		2 wks.	No
		3 wks.	No
Duodenal leak	4	11 days	No
		5 days	No
		40 days	Death
		6 months*	Secondary closure necessary

*This patient had had a previous pyloroplasty. The resection was extremely difficult. The cause for the persistent leakage of bile was attributed to the silk sutures which were used for the duodenal closure at the time of gastrectomy. They were demonstrated when the secondary closure was carried out six months later. For us this case is a unique experience.

for a persistent small leak. Persistence was found to be caused by a silk suture. Severe postoperative gastric atony occurred 3 times and required prolonged gastric drainage by Levine tube. Spontaneous adjustment occurred and there has been no subsequent difficulty.

There were 4 deaths in this series of 187 patients, an operative mortality of 2.1 per cent. The causes of death are indicated in Table VII. It is interesting to note that in the last 116 cases of the series, there was no mortality.

TABLE VI. OPERATIVE MORTALITY

Total number of patients		187
Number of deaths		4
Operative mortality	2.1%	

TABLE VII. CAUSES OF DEATH

CAUSE	NUMBER	AUTOPSY
Pulmonary embolus	1	Yes
Pylephlebitis with multiple liver abscess	1	Yes
Death on operating table (anesthesia death?)	1	No
Duodenal leak occurring 17 days postoperative and resulting in death of patient 40 days later	1	No

FOLLOW-UP

The patients in this series have been followed carefully. With few exceptions, each patient has returned for check-up observation every six months. Inasmuch as the patients in this group were operated upon during the years between 1934 and 1947, the period of observation varies from over thirteen years to six months. We plan to follow the same people as long as possible and it is hoped that, at a later date, we will be able to report the ten-year and later follow-up in each instance. This would be of inestimable value in appraising the long-term efficacy of gastrectomy in the treatment of duodenal ulcer. So far in this carefuly studied group there have been only 4 instances of recurrent ulceration. In 3, a completely adequate gastrectomy was performed and there developed a gastrojejunal ulcer. Each patient was treated during 1948 by vagotomy alone with spontaneous healing of the ulcer.

The fourth patient, already referred to in the early part of the paper, had a prepyloric Finsterer exclusion operation performed in 1940. He returned in

TABLE VIII. DURATION OF FOLLOW-UP OBSERVATION

YEARS	NUMBER OF PATIENTS
1	40
2	27
3	40
4	24
5	20
6	7
7	8
8	10
9	0
10	2
11	1
12	1
Over 12	3

December, 1947, with a gastrojejunocolic fistula which was repaired by a three-stage Pfeiffer operation in April, 1947. He has been well since. If this case is excluded because it was definitely an incomplete gastrectomy originally, there is an incidence of 3 gastrojejunal ulcers in 168 patients, with primary duodenal ulcer an incidence of 1.7 per cent. We are, of course, not including in this particular tabulation the 12 instances of gastrojejunal ulcer and gastrojejunocolic fistula, secondary to gastroenterostomy done elsewhere.

In appraising the patients' complaints at the time of follow-up examinations we have tried to err on the side of magnifying these complaints rather than to minimize them. It has become quite evident that the vast majority of the people in this series have been restored to robust health with normal gastric function after years of digestive invalidism. In our experience this is one of the most satisfying aspects of gastrointestinal surgery. An attempt has been made in Table IX to list the follow-up complaints of some of these patients. In only 3 instances have we encountered the set of symptoms usually described as the "dumping" syndrome. Whether these symptoms are caused by a transitory morning hypoglycemia or an actual rapid emptying of the stomach is still a moot question. Our observations would cause us to lean toward the first hypothesis rather than the second. Curiously enough, after varying periods of time, these symptoms abated completely. And so it has been with most of the complaints listed in Table IX. Those patients who are listed as not having

TABLE IX. FOLLOW-UP STUDIES

SYMPTOMS	NUMBER OF PATIENTS
Symptoms suggesting "dumping" syndrome	3, negative x-ray
Post-cibal nausea	4, negative x-ray
Post-cibal fullness	4, one had negative x-ray
Inability to gain weight	36
Belching and pyrosis	3, negative x-ray
Epigastric distress	4, negative x-ray
Persistent epigastric distress and indigestion (psychoneurosis?)	1, repeated negative x-ray
Anorexia	3
Persistent attacks of diarrhea	2, negative x-ray
Scar pain	3
Intolerance to sweets	12
Intolerance to milk	9

gained weight have persisted in their inability to do so. One patient has persistent epigastric discomfort and indigestion. Repeated gastrointestinal x-ray examinations have been regularly negative. There is alleged to be a strong psychoneurotic element in this patient.

We have tried to have every patient submit to a follow-up test meal examination. Approximately 50 per cent have refused because of their continued good health and absence of any abnormal gastrointestinal symptoms. We have not been too insistent about the necessity for this examination because the continued well-being of the patient is a better barometer of the physiology of his stomach than the results of a test meal might indicate. With very few exceptions, all those patients who have had test meals have shown absent or greatly diminished free HCl. Eighteen patients listed in Table IX have had one or more roentgenographic examinations with negative findings. We have not seen, in this series, a single instance of severe anemia or secondary carcinoma in the gastric stump.

CONCLUSIONS

On the basis of the findings resulting from a careful analysis of 187 patients with duodenal ulcer treated by adequate gastric resection, we have come to the following conclusions:

1. The operation can be carried out with a low operative mortality, 2.1 per cent in this series. There was no mortality among the last 116 cases.

2. The incidence of postoperative duodenal stump leakage can, by the application of basic surgical principles, be reduced to a neglible figure.

3. The posterior gastrojejunal anastomosis gives far fewer postoperative mechanical difficulties.

4. The desired effect following gastrectomy is a persistently maintained absence or marked diminution of free HCl.

5. The follow-up studies in this series indicate a negligible incidence of gastrojejunal ulcer.

6. There is insufficient evidence at this time to warrant the statement that the operation of vagotomy can or should supplant a properly performed gastric resection for duodenal ulcer.

7. There is some evidence to indicate that vagotomy should be utilized as an adjuvant procedure to gastric resection in those cases of duodenal ulcer associated with an abnormal acidity or repeated bleeding episodes.

8. Vagotomy seems to be the operation of choice for the rare instance of gastrojejunal ulcer following a properly performed gastrectomy for duodenal ulcer.

Note.—We wish to emphasize clearly that this paper deals solely with duodenal ulcer. The problem of gastric ulcer is altogether a different one, certainly a simpler one and not concerned with the physiological disturbances accompanying duodenal ulcer. Therefore the lumping together of duodenal and gastric ulcer patients when reporting statistics is to be deprecated.

ADDENDUM

Since this paper was submitted for publication, three patients have died, two of coronary occlusion and one of pneumonia. The deaths were three, five, and seven years, respectively, after gastrectomy.

A COMPARATIVE STUDY OF VARIOUS SUBSTANCES FOR THE PREVENTION OF ADHESIONS ABOUT TENDONS

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THE adherence of tendons at the site of trauma or infection has been and continues to be a difficult problem for the surgeon to overcome. If the scarring in the region of adherence is severe, replacement of the cicatrix by normal soft tissues through which the tendons can move is necessary. This is accomplished either by moving adjacent normal tissue into the area or by means of a pedicle flap. In less severe cases, tendolysis and early motion is of proved value when the tendons are not divided.

A material which will prevent recurrence of adhesions when placed next to tendon is highly desirable. Bunnell[1] recommended the use of thin layers of autogenous fascia or the veil-like material dissected from the outer surface of the fascia lata placed on one side only of the tendon. Cellophane has been employed by Farmer[2] and Wheeldon[3] and tantalum foil by Pearlman,[4] with varying degrees of success.

This study was carried out to test and compare the value of a number of materials in preventing adhesions when placed immediately about injured tendon in the dog. Special interest centered on some of the more recently developed absorbable substances which are now available.

The following materials were studied: (1) autogenous fascia from the outer surface of the fascia lata, (2) human fibrin film,* (3) bovine fibrin film,† (4) Gelfoam,‡ (5) cellophane,§ (300-PUT-71) (6) Oxycel cotton.‖

METHOD

Dogs were used in all experiments. Under intravenous nembutal anesthesia and aseptic conditions the flexor carpi ulnaris tendon of each leg was exposed through a curved lateral incision just above the paw. An Allis forceps was drawn over each tendon eight times to traumatize its surface following which steel wire markers were placed one inch apart under the tendon through adjacent tissues. The experimental material was wrapped about one tendon for one and one-half inches and the steel wire markers tied loosely about tendon and material. The material projected one-fourth inch beyond each wire marker. On the other leg in each animal the steel wire markers were tied loosely about

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*Obtained from Cutter Laboratories, Berkeley, Calif.

†Supplied by The Armour Laboratories, Chicago, Ill.

‡Supplied by The Upjohn Company, Kalamazoo, Mich.

§Supplied by E. I. DuPont de Nemours and Company, Wilmington, Del.

‖Supplied by Parke, Davis & Company, Detroit, Mich.

tendon only. Following this the wounds of each leg were closed by means of wire sutures to the skin. The materials were wrapped completely about the experimental tendons in all but one experiment. In this instance human fibrin film was placed around one tendon and under the other in each animal.

Three to four weeks later the dogs were re-operated upon. The steel wire markers were identified through short incisions proximal and distal to the one-inch areas. The segment of tendon between the wire markers was not disturbed. The portions of tendon lying outside the wire markers were freed by sharp dissection and divided. Hemostats were then applied to the proximal end of each tendon segment and the wire markers removed. The force required to pull the tendon segments free from their beds was then determined by means of a weight and pulley system.

In the experiments using autogenous fascia this material was obtained through a vertical incision on the thigh of each animal. The thin slippery layer of fascia immediately overlying the fascia lata was used. This was kept moist in a saline sponge until placed about the flexor carpi ulnaris tendon a few minutes later. The smooth slippery surface was always placed next to the tendon.

In the earlier studies, the front legs of the animals were immobilized in plaster casts for a maximum of three weeks after the first operation. In later experiments the wounds were covered with dry gauze dressings only. In all instances the two front legs of the animal were treated in identical manner after operation.

AUTOGENOUS FASCIA FROM THE OUTER SURFACE OF THE FASCIA LATA

Eleven animals were prepared with autogenous fascia by the method just described. The tendon segments were pulled free from their beds twenty-nine days later. In each animal the experimental tendon pulled free more easily than did the control tendon. Two of the control tendons in large animals were so firmly adherent that the hemostats pulled off before the tendons pulled free. All of the experimental tendons pulled free. The average force required to pull free the control tendon segments was 4,422 Gm. The eleven tendons in the same animals which were wrapped with autogenous fascia pulled free with an average force of 2,638 Gm. or 60 per cent of the force required for the controls. The probability of a difference as great as this occurring by chance alone is less than one in one hundred times, which is considered significant from a statistical standpoint. This verifies the clinical impression that thin layers of fibroareolar tissue are of value in the prevention of adhesions about tendons. Fig. 1 shows the microscopic appearance of tendon and adjacent tissue in cross section at nineteen days. The fascia is probably represented by the wavy fibrous layer just away from the surface of the tendon. There is connective tissue between tendon and fascia and between fascia and surrounding tissue. There is no line of demarcation between tendon and surrounding tissues.

HUMAN FIBRIN FILM

In this experiment, two series of dogs were prepared. The first series consisted of twelve animals with human fibrin film completely around one flexor

carpi ulnaris tendon and nothing around the other. The tendon segments were pulled free twenty-eight days later. In three of the animals, the tendon was found to be soft and friable, and did not have enough substance to be pulled free. It appeared that the film completely about the tendons in these three animals interfered with the nourishment of the tendon to such an extent that this change occurred. In the other nine animals of this series the experimental

Fig. 1.—Autogenous fascia, nineteen days. Cross section of tendon and adjacent tissues. The fascia is probably represented by the wavy area of fibrous tissues just away from the surface of the tendon. There is no cleavage plane between tendon and adjacent tissues (×86).

tendons were well preserved and pulled free with an average force of 867 Gm. The average force necessary to pull free the control tendon segments was 4,195 Gm., or, in other words, only 21 per cent of the control force was required to pull free the tendon segments which were surrounded by human fibrin film. Some of the experimental tendons were so free from adhesions that a force as little as 200 Gm. was sufficient to dislodge the segment. On the other hand, four of the control tendons were too firmly adherent to be pulled free. A thin layer

of the fibrin film was still identifiable about the tendons at the end of four weeks. This material was lying free in the tissues. Fig. 2 shows the microscopic picture of the tendon at the time it was pulled free on the twenty-eighth day. The layer of fibrin film is not shown, though it was still present at this time. A rather thick zone of young connective tissue is seen next to the tendon. There is a palisading of fibroblasts on the surface resembling epitheloid cells which forms a smooth glossy surface. The fibrin film separated the tendon from adjacent tissues.

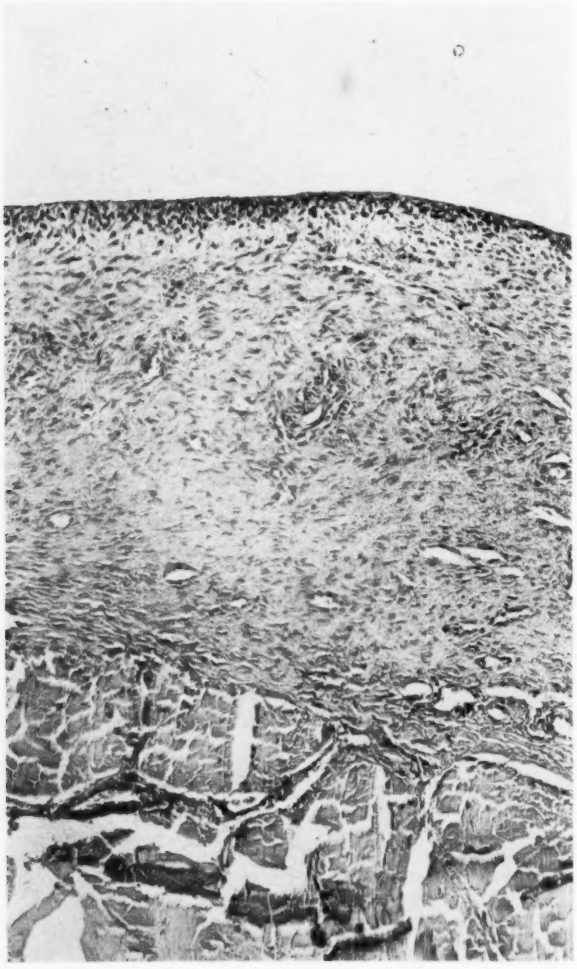

Fig. 2.—Human fibrin film, twenty-eight days. Cross section of edge of tendon which has been surrounded by human fibrin film. Note the smooth surface layer of cells at the upper margin of the section. The film, though not shown, was still present adjacent to this surface layer of cells. The film separated the tendon from adjacent tissue (×86).

In the second series of twelve animals, human fibrin film was placed completely around one flexor carpi ulnaris tendon and merely under the other. These tendon segments were pulled free three weeks later. Two of the twelve tendons in this second series with the film wrapped about them disintegrated, as did three in the first series described. Combining the tendons in both series, five out of twenty-four disintegrated when human fibrin film was wrapped

about them for a distance of one and one-half inches. No such disintegration was observed in the twelve tendons with human fibrin film on one side only, which pulled free with an average force of 2,404 Gm. as compared to 684 Gm. when the material was placed completely around the tendon. It is seen by this that placing film completely around the tendons is more efficient in preventing adhesions, but that it may interfere seriously with the nourishment of the structure.

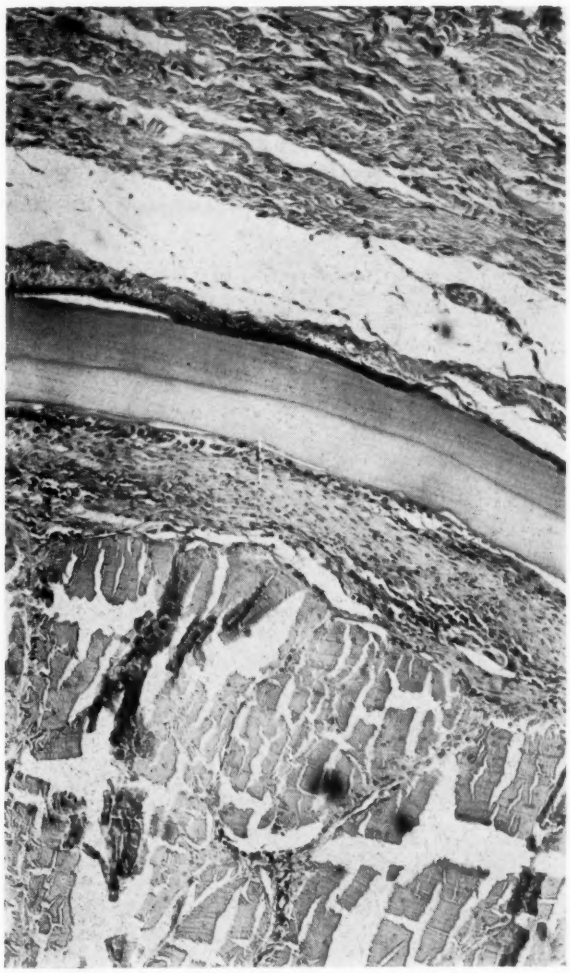

Fig. 3.—Bovine fibrin film, twenty-eight days. Cross section of tendon and adjacent tissues showing the film still in place (×86).

It is of interest to compare the control group of the first series (nothing about the tendons) to the group of the second series which had film placed only on one side. The average force for the former was 4,195 compared to 2,404 Gm. for the latter group. No accurate conclusion can be drawn from this comparison because of the difference of one week in the time interval. It is our opinion, however, that the figures would not be greatly different if the time intervals were identical.

BOVINE FIBRIN FILM

Five animals were prepared with bovine fibrin film around one tendon and nothing on the opposite side. The tendon segments were pulled free four weeks later. The average control figure was 3,940 Gm. and the average experimental figure was 805 Gm., 21 per cent of the control. In this small series, bovine fibrin film appeared to be just as effective as human fibrin film. There is still some question as to whether this material might be antigenic when used in man and its clinical use should not be made until this is settled. Histologic reaction to this material is shown in Fig. 3. In other areas the zone of connective tissue reaction was wider than shown in this photomicrograph. It is our impression that the reaction about the human and bovine fibrin films was about equal in our animals.

GELFOAM

Five animals were used in this group with Gelfoam. After a four-week interval, the average force necessary to pull free the control segments was 3,470

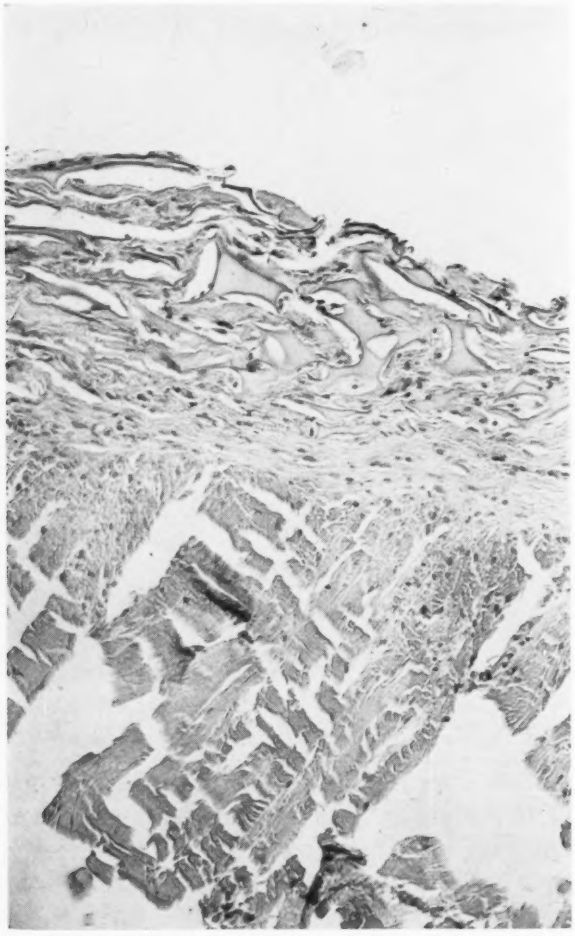

Fig. 4.—Gelfoam, twenty-eight days. Cross section of edge of tendon with Gelfoam still present on its surface. Note that fibrous tissue has grown into the interstices of the material (×150).

Gm., and the average force for the experimental tendon segments was 2,160 Gm., or 62 per cent of the average control figure.

The probability of these results occurring by chance alone is three times in one hundred which is still considered significant from a statistical standpoint.

Fig. 4 shows the reaction of the tissues to this material to be relatively small. It is seen that the fibrous connective tissue had grown into the interstices of the material. There is no free cleavage plane as with the film materials.

OXYCEL COTTON

In six animals Oxycel cotton was placed completely around one tendon and nothing about the other. The tendons were pulled free twenty-seven days later. In two of the animals the experimental tendons were soft and friable due to interference with nourishment. The average force for the controls was 4,458 Gm., compared to 3,913 Gm. for the experimental tendons. This is 88 per cent

Fig. 5.—Cellophane, twenty-six days. Cross section of edge of tendon surrounded by cellophane which is seen at the upper edge of the section. Note the palisading of the surface layer of cells next to the material (×86).

of the control force and, in our opinion, indicates that the material has little or no value in preventing adhesions about injured tendons. The difference is not statistically significant as judged by the t test: $t = 1.01$ $P = .35$.

CELLOPHANE 300-PUT-71

A single thickness of cellophane 300-PUT-71 was placed completely around one flexor carpi ulnaris tendon and nothing about the other tendon in six animals. The tendon segments were pulled free twenty-seven days later. The average force for the controls was 4,041 Gm., and for the experimental tendons 950 Gm., 24 per cent of the control figure. One tendon of the six disintegrated due to lack of nourishment with the material completely surrounding it. The difference between controls and experimental forces is statistically significant as shown by the t test. $t = 10.23$ $P = < .01$.

Fig. 5 shows the reaction to this material. The film is seen apparently unchanged twenty-six days after being placed about the tendon. A rather thick zone of connective tissue is seen next to the tendon on the surface of which there is palisading of fibroblasts forming a smooth surface. This appearance is comparable to that recently reported by Wilson[5] and to that seen with human fibrin film (Fig. 2).

DISCUSSION

The film materials were most effective in preventing adherence of the tendons. Human fibrin film, bovine fibrin film, and cellophane were about equally effective, all three being more effective than thin layers of autogenous fascia. However, the reaction of the tissues about the blood fraction films and the cellophane was greater than that about the autogenous fascia. The smooth surface layer of cells next to human fibrin film and cellophane is interesting. This surface layer of cells resembling synovia was not seen on the surface of the tendons wrapped with fascia.

Gelfoam, though it had small reaction in the tissues, was not so effective as human or bovine fibrin film or cellophane, due probably to the fact that the tissues grew into the interstices of the material.

The fact that five out of twenty-four tendons completely surrounded by human fibrin film and one of six tendons surrounded by cellophane showed disintegration indicates that these materials at times seriously interfere with the nourishment of the tendon when thus applied. It would seem wise not to apply these materials completely about tendons but rather to use them on one side only as recommended by Bunnell for autogenous fascia. The results with a perforated film have not been investigated. It is of interest that none of the tendons wrapped with thin layers of autogenous fascia over a one and one-half inch distance showed disintegration. Possibly this might have occurred if a greater length of tendon had been covered. Until the antigenic properties of bovine fibrin film are completely worked out it would seem that clinical use of this material should be withheld.

SUMMARY

Six materials were tested for their effectiveness in preventing adhesions when placed next to injured tendon in the dog. The results are summarized in Table I.

TABLE I

MATERIAL	NUMBER OF DOGS	AVERAGE GRAMS		PER CENT
		EXPERIMENTAL	CONTROL	
Human fibrin film	12	867	4195	21
Bovine fibrin film	5	805	3940	21
Cellophane	6	950	4041	24
Autogenous fascia	11	2638	4422	60
Gelfoam	5	2160	3470	62
Oxycel cotton	6	3912	4458	88

The average force required to pull free the experimental and control segments of tendons is given in columns three and four. The last column gives the per cent of the control force required to free the experimental segments for each material.

Human fibrin film and bovine fibrin film were still present, usually thinned out, and cellophane apparently was unchanged at the end of four weeks. In some instances bovine fibrin film was apparently unchanged in thickness at the end of four weeks.

Fibrin film of bovine or human origin as well as cellophane each produced a zone of vascular connective tissue about the tendon in the dog. Many instances were seen where a surface layer of cells resembling synovia had formed on the surface of this zone of reaction.

In five of twenty-four tendons wrapped with human fibrin film over a one and one-half inch distance and in one of the six wrapped with cellophane, disintegration of the tendons occurred apparently due to interference with nutrition of the tendon. Such changes were not seen in the controls or when human fibrin film was placed on one side only of the tendon in twelve animals.

CONCLUSIONS

1. Human fibrin film, bovine fibrin film, and cellophane were most effective of the materials tested in preventing adhesions about injured tendons in the dog.
2. Autogenous fascia was of definite value but less than those just listed.
3. Film materials may seriously interfere with the nourishment of tendons if wrapped about them.

Appreciation is expressed to Dr. Carl H. Lenhart for his helpful interest in this work and to Dr. Herbert Z. Lund and the staff of the Institute of Pathology for aid with microscopic preparations and photomicrographs.

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RUPTURES OF MUSCLES AND TENDONS

With Particular Reference to Rupture (or Elongation of Long Tendon) of Biceps Brachii With Report of Fifty Cases

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IN A previous communication one of us (R. L. W.) discussed rupture of the biceps brachii based on a study of 14 patients personally observed and treated over a period of eight years.

Since that time, during a period of a little over six years, we have encountered an additional 36 cases. This number, together with the 14 cases previously reported, makes a total of 50 cases of rupture of the biceps brachii personally observed and treated during a total period of a little over fourteen years. The 50 cases were found in a total number of 60,792 hospital admissions. This group of cases, however, does not include several other patients with this condition seen by the senior author during the past several years who were under treatment by our colleagues in other hospitals at the time.

The more frequent recognition of this interesting musculoskeletal lesion, during the past few years has been due, no doubt, to the continued "biceps mindedness" in the diagnosis of shoulder conditions by our resident staff.

Although rupture of the biceps brachii is not unusual, the condition all too frequently goes unrecognized and untreated because the physician or surgeon is not familiar with the condition. This is illustrated in several of our cases in which the diagnosis on admission to hospital was recorded as "strain or sprain of the shoulder," "subdeltoid bursitis," "arthritis," "traumatic periarthritis," "rupture or tear of the supraspinatus tendon," or "neuritis." Also, on account of the ecchymosis, which is frequently an accompanying finding in these ruptures, there was a tendency in some instances to consider the primary condition to be a rupture of one of the larger blood vessels.

LITERATURE

The earliest reports of ruptures and tears of muscles and tendons are found in the European literature and date back to the first quarter of the eighteenth century. The first report found in the literature of a rupture of a muscle or tendon, that of a rupture of the Achilles tendon, was by Petit in 1722. The earliest known reference to a rupture of (1) long tendon of the biceps brachii was by Chamseru in 1761, and (2) a rupture of distal tendon of biceps brachii by Acquaviva in 1898. The first to report a rupture of the supraspinatus tendon was Smith in 1835. Since then there have been many other reports, so now

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there may be found reports of ruptures of nearly all of the more important muscles and tendons. While there has been an increasing number of contributions relative to rupture of the biceps brachii, during the past two decades the most outstanding have been those by Gilcreest.

GROSS ANATOMY

The long head of the biceps brachii arises by a round tendon from the supraglenoid tubercle at the apex of the glenoid cavity and then becomes flattened as it passes through the cavity of the shoulder joint. It emerges as a round tendon again, through an opening in the capsular ligament, and beneath the transverse ligament it occupies the bicipital groove of the humerus (sulcus intertubercularis). It is held in the bicipital groove by the transverse humeral ligament and the fibrous expansion from the pectoralis major muscle.

The short head, in common with the coracobrachialis, arises from the tip of the coracoid process of the scapula. Tendinous only at first, it forms a separate fleshy belly, which is united to the long head by an investment of the deep fascia.

The insertion of the biceps brachii is likewise twofold. The two bellies become connected with a strong tendon attached deeply in the hollow of the elbow to the tuberosity of the radius and by a strong membranous band (lacertus fibrosus) to join the deep fascia covering the origins of the flexor and pronator muscles of the forearm.

The biceps brachii is an extremely variable muscle. Its chief anomalies are due to an increase in the number of origins and insertions. A third head is common (10 per cent) and usually arises from the humerus or from the bicipital groove. The muscle may also have an additional insertion into the medial condyle of the humerus or into the fascia of the forearm.

The nerve supply to the biceps brachii is by the musculocutaneous nerve (C. 5, 6). The action of the biceps brachii is to supinate the forearm, flex the elbow, and with the aid of the coracobrachialis to adduct and flex the humerus at the shoulder joint.

OCCURRENCE

A review of the literature indicates that the muscles and tendons most frequently ruptured and in order of frequency are: muscles of the calf extensors of the leg, biceps brachii, Achilles tendon, extensors of the thumb and fingers, supraspinatus tendon, rectus abdominis, adductors of the thigh, triceps brachii, and pectoral muscles. However, in our experience the order of frequency of rupture of muscles and tendons has been biceps brachii, muscles of the calf, Achilles tendon, supraspinatus tendon, extensors of the leg, extensors of the thumb and fingers, and triceps brachii. We have not thus far encountered ruptures of the rectus abdominis, the adductors of the thigh, or the pectoral muscles.

Codman, reporting 500 cases of shoulder injuries, found 122 cases of rupture of the supraspinatus and only 9 cases of rupture of the biceps. He found that rupture of the biceps is not infrequently associated with tears of the supraspinatus tendon, and was of the opinion that this fact frequently misled

many surgeons to attribute supraspinatus tendon symptoms to those due to the associated biceps rupture. He believed that rupture of the long tendon of the biceps was a trivial matter, but when combined with an injury to the supraspinatus became very serious because of the help of the long tendon in maintaining the head of the humerus against the glenoid cavity.

Biancheri reported that ruptures of the biceps brachii are more frequent in the long head and its tendon. He found that the long head and its tendon was affected in 96 per cent of cases, the distal tendon in 3 per cent, and the short head in only 1 per cent of cases.

In our series of 50 cases, there were 45, or 90 per cent, involving the long head and its tendon and 5, or 10 per cent, involving the distal tendon. These 50 cases were found in a total of 60,792 hospital admissions or 1 case in every 1,200 admissions to the hospital.

Of the 50 cases of rupture of the biceps brachii, 30 were of the right arm and 20 of the left arm, while 6 (3 patients) were bilateral lesions. Only one of the patients with a rupture of the biceps of the left arm was left-handed. That the right arm is more often affected than the left may be explained by the fact that the great majority of patients are right-handed and that the right arm is more often exposed to indirect forms of injury.

The oldest patient with rupture of the biceps brachii in this series of 50 cases was 74 years and the youngest was 32 years, while the average was 54 years of age.

MECHANISM OF RUPTURE

In considering ruptures of muscles and tendons one should understand the mechanism involved in the production of these injuries. Ruptures of muscles and tendons are more commonly the result of muscular action, particularly when the muscle is subjected to forceful contraction. External trauma and spiral twists or torsions of the limb may also produce ruptures of muscles and tendons.

The sudden contraction of the biceps brachii sufficient to rupture the tendon or muscle, more frequently occurs as a result of sudden or uncoordinated movements such as lifting while in an awkward or strained position. Gilcreest, however, stated that a fall on the shoulder, usually considered a form of direct trauma, may throw a great strain on the long tendon and in this way may indirectly produce such a rupture.

Degenerative and pathologic changes in the muscle or tendon present at the time of injury may be predisposing causes. Meyer in the dissecting room found many anatomic specimens of the shoulder and arm, in which there were atrophy of the muscles and varying degrees of destruction of the tendon of the long head of the biceps, due apparently to "wear and tear" or attrition of the tendon. He stated that in cases of long-standing rupture of the supraspinatus tendon, the intra-articular portion of the biceps tendon is often absent. When the attachment at the supraglenoid tubercle is lost, the tendon of the long head may become reattached in the bicipital groove or to the transverse humeral ligament. At times, the intracapsular portion of the long tendon may disappear completely

or may fuse with the joint capsule. Meyer also stated that rupture of the long tendon may occur with an intact shoulder capsule.

Bouchers made a very careful study of the mechanical factors involved in ruptures of the long tendon. He concluded that ruptures above the bicipital groove should be attributed to muscular effort alone but if there be present in the tendon or its sheath any condition which would impede the free passage of the tendon through the intertubercular notch, that condition would be favorable to rupture and should be accorded recognition as a predisposing factor.

According to Bouchers, osteophytic outgrowths appear on the tuberosities of the humerus and grow toward each other, thus tending to convert the groove into a canal. The same process takes place in the floor of the groove and along the bicipital ridges. These outgrowths are at first smooth, rounded excrescences; later they add a sharply pointed top or spicule. As a result of the outgrowths the groove may be converted into a canal and the tendon imprisoned therein; the groove may be filled in and the tendon be dislodged mechanically from its bed, or the early formation of spicules may cause the tendon to chafe itself incidental to its back-and-forth motion in the groove.

It is difficult to postulate as to the mechanism of a rupture occurring at a musculotendinous junction. Ludington, however, was emphatic in stating that all ruptures occurring outside or below the bicipital groove are of traumatic origin. Ludington referred to 2 cases in which the rupture was below the groove. In one of these cases, the patient had grasped a rapidly revolving flywheel, with a resultant rupture at the junction of the tendon and belly of the long head. He assumed that the maximum tension came on the long head with the forearm quite extended and the humerus so drawn away from the body as to permit the long head to pass from its insertion to its origin in a straight line, instead of lying in and following the curve of the humeral head as would usually be the case in lifting. He concluded, therefore, that the rupture occurred at the recognized weak point of the flexor mechanism, that is, the junction of the tendon and muscle belly, because the mechanical conditions were such as to equalize the distribution of strain. In Ludington's second case of rupture below the groove, there was a history of repeated direct trauma to the tendon at the site of its rupture and at operation "a fusiform ecchymotic swelling was found" which was believed to have diminished its tensile strength.

Based on a study of our cases we are in agreement with Ludington's explanation as to the probable mechanism of the injury in cases of rupture at the musculotendinous junction, since a study of our cases shows that injury was either of a direct type or had probably occurred with the arm extended as would be expected in checking a fall by grasping the rung of a ladder.

In the production of a rupture of a muscle or tendon, occupation plays an important role. Gilcreest stated that ruptures of the neck muscles usually occur in hod carriers and packers; of the biceps and triceps in lifters and baseball pitchers; of the adductors of the thigh in horseback riders; of the calf muscles in boxers, tennis players, runners, and mountain climbers; of the extensor pollicis in drum players; and of the back muscles in stevedores.

TABLE I. SUMMARY OF CASES; RUPTURE OF BICEPS BRACHII

CASE	AGE (YR.)	WORK	TIME LAPSE SINCE INJURY	INJURY	SIDE AFFECTED	SYMPTOMS	SIGNS	OPERATIVE FINDINGS	NATURE OF OPERATION	RESULT
15*	41	Carpenter	4 yr.	Fall, striking shoulder	R	Pain, weakness, bunching	Muscle deformity	Long tendon atrophied and lax	Tendon divided at musculotendinous junction; long tendon threaded through belly of muscle and sutured to muscle and fascia, silk sutures	Good
16	46	Laborer	5 days	Lifting 80 lb. weight	L	Pain, bunching	Muscle deformity, ecchymosis, sulcus	Rupture of tendon near glenoid end coiled	Tendon woven through short head and sutured to coracoid process	Good
17	46	Laborer	2 yr.	Lifting heavy object	R	Pain, weakness	Sulcus, muscle deformity, lessened power, tenderness	Tendon relaxed and elongated 1 inch, frayed out but not completely divided	Tendon detached high, passed through short head and sutured through drill hole in coracoid process, silk sutures	Good
18	67	Deckhand	3 mo.	Throwing a heaving line	R	History of ecchymosis, pain, weakness	Muscle deformity, lessened power, sulcus	Lateral portion of muscle displaced distally; only sheath of tendon left, long tendon folded on self	Tendon attached to humerus through drill hole and sutured back on self, silk sutures	Good
19	57	Pipelayer	6 wk.	Hit on shoulder by falling pipe	R	Pain, weakness	Muscle deformity, lessened power	No operation	No operation	No operation

*Cases 1 to 14, inclusive, have been previously reported.[46]

20	41	Laborer	3 wk.	Fell 12 feet, landing on shoulder	R	Weakness, bunching	Muscle deformity, sulcus	No operation	No operation	No operation
21	46	Engineer	5 mo.	Pulling on a line	L	Pain, weakness	Muscle deformity, sulcus distal to belly	Scarring of distal tendon with adhesions about lacertus fibrosus	Scarred tendon dissected and sutured to tendinous portion of brachialis anticus by fascial transplant; suture of lacertus fibrosus, cotton sutures	Good
22	61	Oiler	7 wk.	Forcibly thrown to floor	R	Pain, weakness, bunching	Muscle deformity	Cystic degeneration of elongated tendon; tendon had lumen	Tendon separated in bicipital groove and fixed by loop around insertion of deltoid muscle and sutured back on self, cotton sutures	Good
23	61	Oiler	7 wk.	Forcibly thrown to floor	L	Pain, weakness, bunching	Muscle deformity, tenderness	Cystic degeneration of elongated tendon; tendon had lumen	Tendon separated in bicipital groove and fixed by loop around insertion of deltoid muscle and sutured back on self, cotton sutures	Good
24	58	Elevator operator	6 mo.	Strained arm and received sudden jolt	L	Pain, weakness, bunching	Muscle deformity	Long tendon slack and belly displaced distally; excess scar tissue in upper portion of belly at musculotendinous junction	Long tendon sectioned and free end fixed to attachment of pectoralis major, cotton sutures	Good

(Continued on following pages)

TABLE I.—CONT'D

CASE	AGE (YR.)	WORK	TIME LAPSE SINCE INJURY	INJURY	SIDE AFFECTED	SYMPTOMS	SIGNS	OPERATIVE FINDINGS	NATURE OF OPERATION	RESULT
25	58	Elevator operator	6 mo.	Pushing cart off elevator	R	Weakness, bunching	Muscle deformity	Long tendon torn, atrophic, and frayed proximally; torn ends of tendon fixed by scar tissue and peritendinous adhesions	Long tendon freed and sectioned as high as possible; long tendon then hooked through insertion fibers of pectoralis major and tendon doubled back upon itself and sutured, cotton sutures	Good
26	73	Laborer	6 yr.	Lifting heavy object	R	Bunching, weakness	Muscle deformity	No operation	No operation	No operation
27	57	Deckhand	14 yr.	Pulling on rope	R	Pain, bunching	Muscle deformity, discomfort	No operation	No operation	No operation
28	52	Seaman	17 yr.	Pulling on line	R	Weakness, bunching	Muscle deformity	No operation	No operation	No operation
29	52	Seaman	11 yr.	Fall	L	Weakness, bunching	Muscle deformity	No operation	No operation	No operation
30	62	Laborer	40 yr.	Throwing baseball	R	Pain	Muscle deformity, weakness	No operation	No operation	No operation
31	54	Machinist	5 wk.	Fell catching weight on right arm	R	Pain, weakness	Muscle deformity, weakness	The belly was displaced proximally; distal tendon avulsed from radial tuberosity, distal tendon loosely scarred to lacertus fibrosus	Scar tissue excised; end of biceps tendon split, one-half attached to lacertus fibrosus and remaining portion fixed to radial tuberosity, silk sutures	Good

32	54	Laborer	8 mo.	Lifting object above head	R	History of ecchymosis, pain	Muscle deformity, weakness	No operation	No operation	No operation
33	51	Laborer	3 yr.	Lifting heavy object	L	Pain on lifting	Muscle deformity, sulcus, weakness	No operation	No operation	No operation
34	61	Laborer	Undetermined	Lifting 150 lb.	R	Pain, weakness	Tenderness, lessened power	No operation	No operation	No operation
35	62	Laborer	1 wk.	Pulling lumber	R	History of snapping sensation, pain	Muscle deformity, weakness	No operation	No operation	No operation
36	66	Laborer	2 mo.	Fell down stairway	L	Pain, weakness, numbness	Muscle deformity, tenderness	No operation	No operation	No operation
37	55	Laborer	3 yr.	Lifting heavy object	L	History of snapping sensation	Muscle deformity and lessened power	No operation	No operation	No operation
38	61	Laborer	6 hr.	Lifting heavy object	R	Pain, weakness	Muscle deformity, tenderness, sulcus	No operation	No operation	No operation
39	43	Machinist	9 days	Slipped catching rung of ladder	R	History of ecchymosis, pain, bunching	Muscle deformity, ecchymosis	Torn upper musculotendinous junction with edema, discoloration, organization, and adhesions; *long tendon $\frac{1}{3}$ normal diameter	Long tendon sectioned and fixed to insertion fibers of pectoralis major, cotton sutures	
40	32	Shipfitter	13 days	Fell catching girder	R	Pain, numbness of thumb and index finger	Muscle deformity, tenderness, sulcus	No operation	No operation	No operation

(Continued on following pages)

TABLE I.—CONT'D

CASE	AGE (YR.)	WORK	TIME LAPSE SINCE INJURY	INJURY	SIDE AFFECTED	SIGNS	SYMPTOMS	OPERATIVE FINDINGS	NATURE OF OPERATION	RESULT
41	48	Joiner	3 mo.	Lifting keg of nails	L	Pain when lifting	Muscle deformity, sulcus	Tendon slack; partial rupture with distal displacement of anterior $\frac{2}{3}$ of belly, apparent elongation with apparent displacement of belly	Long tendon sectioned deep in muscle belly; muscle then brought up and threaded over cut tendon, muscle fixed to tendon correcting deformity, cotton sutures	
42	74	Engineer	10 yr.	Operating reverse gear	R	History of pain on lifting for several months, also ecchymosis and bunching	Muscle deformity, tenderness	No operation	No operation	No operation
43	65	Laborer	2 days	Fell on ice	L	Pain, weakness, history of snapping sensation	Muscle deformity, sulcus	Long tendon small and scarred; partial rupture at upper musculotendinous junction	Long tendon freed and sectioned at upper musculotendinous junction; free end carried through muscle and sutured so that muscle and tendon assumed normal position	Good
44	70	Joiner	7 mo.	Lifting heavy box	L	History of snapping sensation, marked weakness, bunching	Muscle deformity, tenderness, sulcus	No operation	No operation	No operation

45	44	Machinist	2 days	Fell catching to ladder	R	Bunching high on arm, pain weakness	Ecchymosis, sulcus distal to muscle belly	Subcutaneous ecchymosis, blood under fascia; distal tendon completely detached	Distal tendon split and sutured to insertion of brachialis anticus and lacertus fibrosus, cotton sutures	Good
46	69	Inspector	5 days	Lifting a 40 lb. bag	L	Discomfort, bunching, weakness	Muscle deformity, sulcus, ecchymosis, tenderness	Long tendon completely divided and lax in sheath; thin gelatinous material in tendon	Tendon sutured to insertion fibers of pectoralis major, cotton sutures	Good
47	60	Joiner	24 days	Lifting	R	Pain, bunching	Muscle deformity, sulcus	No operation	No operation	No operation
48	65	Laborer	19 days	Pushing lumber	R	Discomfort, bunching, weakness	Muscle deformity, sulcus	No operation	No operation	No operation
49	62	Laborer	1 wk.	Lifting heavy object	R	Weakness, bunching, history of snapping sensation	Sulcus, tenderness, ecchymosis; irregularity palpable over proximal part of bunched biceps	Complete rupture of long tendon; end of tendon free in bicipital groove	End of long tendon looped under insertion fibers of pectoralis major; end of tendon then brought down parallel to rest of tendon and free end implanted into muscle belly, cotton sutures	Good
50	47	Electrician	8 days	Pushing 150 lb. weight (storage battery)	R	History of snapping sensation, pain, weakness, and lump in arm	Muscle deformity, sulcus, tenderness, and lessened power	Complete rupture of long tendon high in groove or at glenoid—end of tendon bulbous	Bulbous end resected—tendon looped through expansion of pectoralis major and doubled back and end embedded in belly of muscle, cotton sutures	Good

It is obvious, therefore, that laborers and others necessarily exposed to heavy stress and strain, as in lifting, are as a group more likely to sustain ruptures and tears of muscles, particularly rupture of the long tendon and less frequently the distal tendon of the biceps brachii.

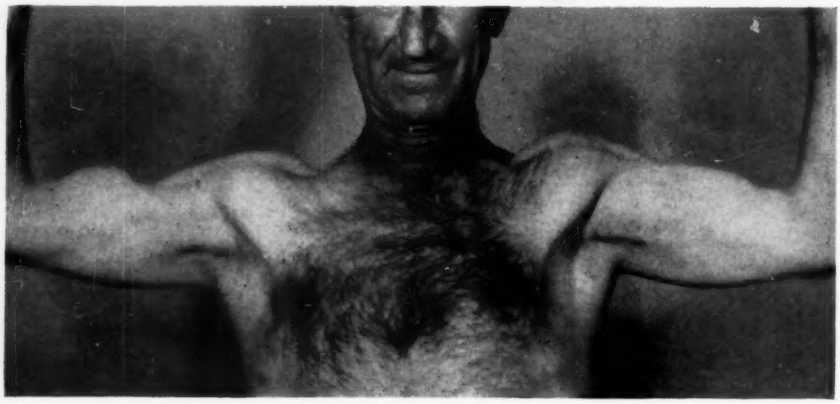

Fig. 1.—Showing rupture of the long tendon of the biceps brachii.

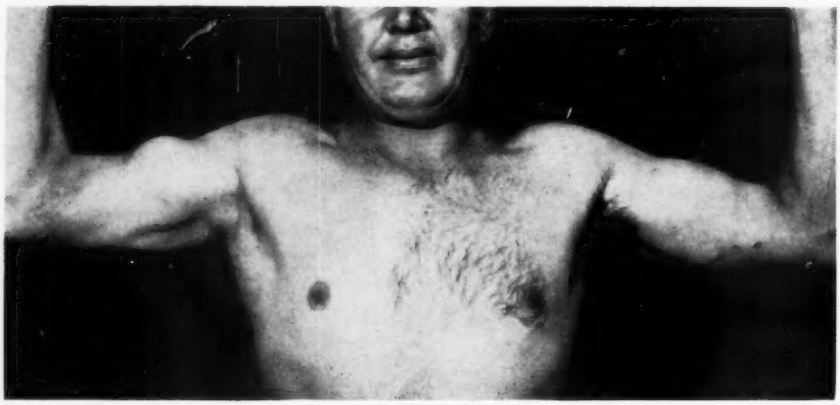

Fig. 2.—Showing rupture (avulsion) of the distal tendon of the biceps brachii.

Gilcreest also stated that excessive fatigue may render muscle fibers less likely to resist a sudden forceful pull or jerk and that this fatigue superimposed upon other predisposing factors may cause rupture more easily. He cited the case reported by Loos, of a man 70 years of age, who, upon returning home after long and wearisome work, raised his arm to take off his hat and in so doing ruptured the long tendon on the left biceps. In this connection one of Harris's four cases of rupture of the long head of the biceps brachialis is of much interest. In this case, the patient was "a hale and hearty old gentleman of 92 years who sustained a rupture of his biceps as a result of suddenly reaching for something above his head."

Gilcreest also mentioned the case of the immortal and versatile John Hunter who sustained a rupture of one of the gastrocnemius muscles while dancing. In discussing the mechanisms of a ruptured muscle, he quoted Hunter in part: "A

broken tendon usually arises from the power of a muscle being put into full action and a sudden resistance taking place at the same time.'' Platt, in his observation of some tendon ruptures, stated that the Edinburgh anatomist and surgeon, Monro (1781), like John Hunter, was himself a victim of this accident. In this connection, there might also be added the name of the late Charles H. Mayo, who, while running upstairs two steps at a time, slipped and fell forward, rupturing a soleus muscle.

SITES OF RUPTURE

The biceps brachii may rupture at almost any site, depending upon the nature of the force of injury and the presence of degenerative changes in the muscle and tendon.

Because a part of the tendon passes through the shoulder joint and bicipital groove, the long tendon of the biceps brachii is exposed to a greater amount of friction or ''wear and tear'' than other tendons. Consequently, a gradual elongation or ''stretching'' or even a rupture of the long tendon may occur. Some partial or incomplete ruptures may be preceded by a gradual fraying out of the long tendon in the bicipital groove followed by adhesions sufficiently strong to hold the tendon but resulting in the usual bicipital syndrome.

In rupture of the long tendon, there are two types, the high and the low. In the high type, the site of rupture is usually at or near the supraglenoid point of origin. In the low type, the rupture is usually found at or slightly below the level of the bicipital groove. It is in some of these cases that the long tendon may be found elongated or slackened but the end fixed by adhesions in the bicipital groove.

The long tendon of the biceps may also be found elongated with obvious atrophic or degenerative changes present but with no surrounding adhesions. The condition (elongation of the long tendon) may exist without a history of previous injury and causing little or no incapacity for work. On the other hand, secondary rupture may occur in an elongated tendon.

Rupture of the biceps brachii may also occur at either the lower or upper musculotendinous junction and may be the result of either direct or indirect violence.

In some instances, in which the long tendon at operation was found to be intact but elongated, there were associated findings indicating either recent trauma (edema and extravasation of blood) or an older injury (scar tissue and adhesions about the upper musculotendinous junction). These findings were believed due to the long tendon having been partially pulled out of its muscle belly.

The distal tendon, although larger and stronger than the long tendon and not subjected to as much ''wear and tear,'' may rupture under certain stresses and strains. In ruptures of the distal tendon, the site is usually at or near its insertion on the tuberosity of the radius.

CLINICAL FEATURES

The symptoms and signs of rupture of the biceps brachii are fairly constant. Most of our patients gave a history of a sudden ''snapping'' sound or sensation,

occurring in the arm or shoulder while in the act of lifting or straining. This was followed by discomfort or aching pain in the arm and shoulder, "bunching" of the muscle, and weakness of the arm. In a few instances, the patient was unaware of the condition except for the bunching deformity of the muscle and lessened muscle power of the arm and shoulder. Several patients noticed areas of ecchymosis over the arm, occuring usually soon after the injury but in some instances delayed for twenty-four to forty-eight hours.

Fig. 3.—Showing a large lipoma simulating in appearance a rupture of the long tendon of the biceps brachii.

Fig. 4.—Showing deformity resulting from paralysis of the biceps brachii (musculocutaneous nerve).

The most common finding is the muscle deformity, essentially a bunching of the belly of the biceps. The bunching is marked if the rupture of the long tendon is complete or if the incomplete or partial rupture takes place at the upper musculotendinous junction. In some patients with complete rupture of

the long tendon there was found an irregularity in contour (both visible and palpable) of the proximal portion of the bunched muscle belly, due to the "curled" or "jack-knived" position of the loosened long tendon.

The site of the bunching deformity of the belly of the biceps with respect to the arm varies with the location of the rupture. In ruptures of the long tendon, the bunching or displaced muscle belly is found nearer the elbow, and in ruptures of the distal tendon the bunching or displaced muscle belly is nearer the shoulder. Also, in these lesions of the long tendon there is a hollowing or sulcus between the upper portion of the belly of the biceps and the deltoid muscle; while in lesions of the distal tendon there is a hollowing or sulcus between the lower portion of the belly of the biceps and the flexed forearm.

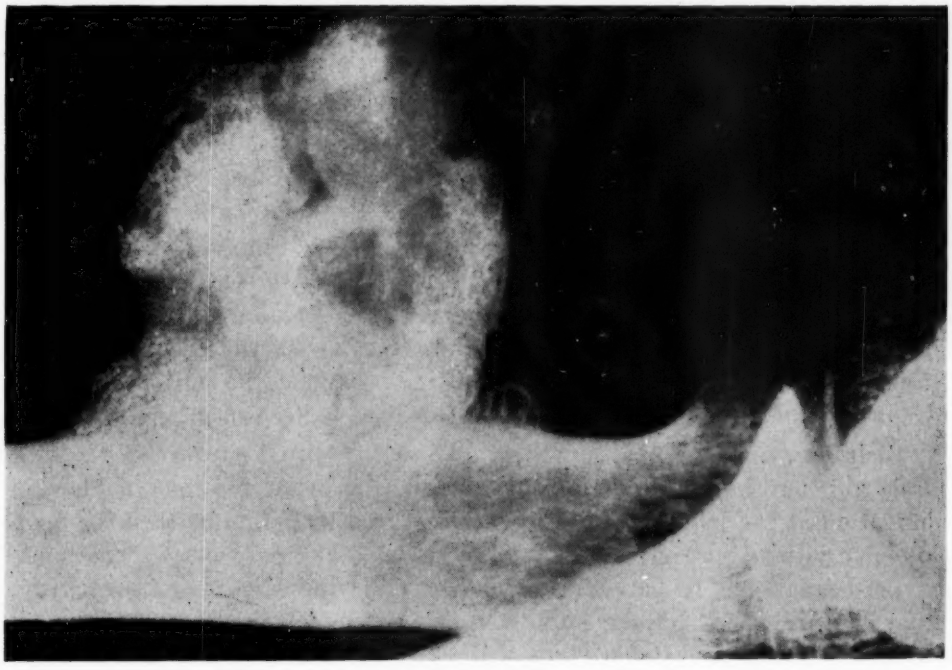

Fig. 5.—Showing an osteoma (myositis ossificans) developing after drilling of the radius in the repair of the rupture (avulsion) of the distal tendon of the biceps brachii.

In patients with heavy muscles, the bunching deformity can be demonstrated more clearly by comparing this region with that of the fellow arm. Therefore, it is very important to examine both arms routinely in all patients in which this condition is suspected.

In patients with rupture of the long tendon, local tenderness is frequently present in the region of the bicipital groove. Loss of function of the arm and shoulder varies from a slight weakness to a moderate loss of power of the arm, more noticeable with the arm in an elevated or abducted position.

DIAGNOSIS

The diagnosis of rupture of the biceps brachii is not difficult if the condition is kept in mind and if, during the examination, the fellow arm and shoulder are exposed and used for comparison.

Heuter, Ludington, and Yergason have described certain tests or maneuvers which are sometimes helpful in the diagnosis of rupture of the long tendon of the biceps brachii.

Heuter's Sign.—Flexion of the forearm in pronation when the biceps is tense is more forcible than when the forearm is supinated and the biceps relaxed.

Ludington's Sign.—The patient is directed to rest his folded hands, palms down, on the top of his head and allow the interlocked fingers to support the weight of the arms. In this position there is maximum relaxation of the long head. The examiner then places two fingers on the tendon of the long head of the biceps, and directs the patient to contract and relax the biceps muscle simultaneously. The contraction of the tendon of the long head on the sound side is felt plainly, while it is absent on the affected side if the tendon is ruptured or is felt less readily if the tendon is elongated.

Yergason's Sign.—If the elbow is flexed to 90 degrees, the forearm being pronated, and the examiner holds the patient's wrist so as to resist supination and then directs that active supination be made against his resistance, pain, very definitely localized in the bicipital groove, indicates a condition of wear and tear of the long head of the biceps or synovitis of its tendon sheath.

In the differential diagnosis one should consider hematoma, herniation of muscle, rupture of the supraspinatus tendon, tumors, both benign and malignant, and inflammatory swellings, that is, abscess. Biceps muscle (musculocutaneous nerve) paralysis and snapping shoulder due to the short head of the biceps muscle slipping over the prominent lesser tuberosity of the humerus, although infrequent, are other conditions one might do well to keep in mind.

Bicipital syndromes may not always be due to actual rupture of the biceps muscle but may be the result of a simple elongation of the long tendon or a subluxation of the long tendon from its groove. Elongation of the long tendon may exist without history of injury and the bunching deformity of the muscle may not be marked. In patients with elongation of the long tendon even without actual rupture, there may be found the characteristic sulcus between the belly of the biceps and the distal portion of the deltoid muscle. Also, there is usually local tenderness in the region of the bicipital groove. Subluxation of the tendon of the biceps from its groove is usually a difficult diagnosis to make, but in this condition there is fairly constant tenderness in the region of the bicipital groove and the long tendon may be palpated as it slips out and in from its groove upon external and internal rotation of the shoulder. The only case of subluxation (dislocation) of the long tendon that we have encountered (but which is not included in this series of cases) is one in which a diagnosis of rupture of the supraspinatus tendon was made, but at operation the long tendon was found completely dislocated from its groove and both the supraspinatus and the long bicipital tendons were found intact.

TREATMENT

The character of treatment should depend upon the degree and site of the rupture, the age of the patient, the presence of coexisting disease, and whether

or not the condition is sufficiently disabling for the kind of work the patient may have to do in the future. If the rupture is partial with little deformity and only slight loss of function, good results should be expected from conservative measures.

However, in a considerable number of patients operative repair of the ruptured muscle or tendon is the treatment of choice.

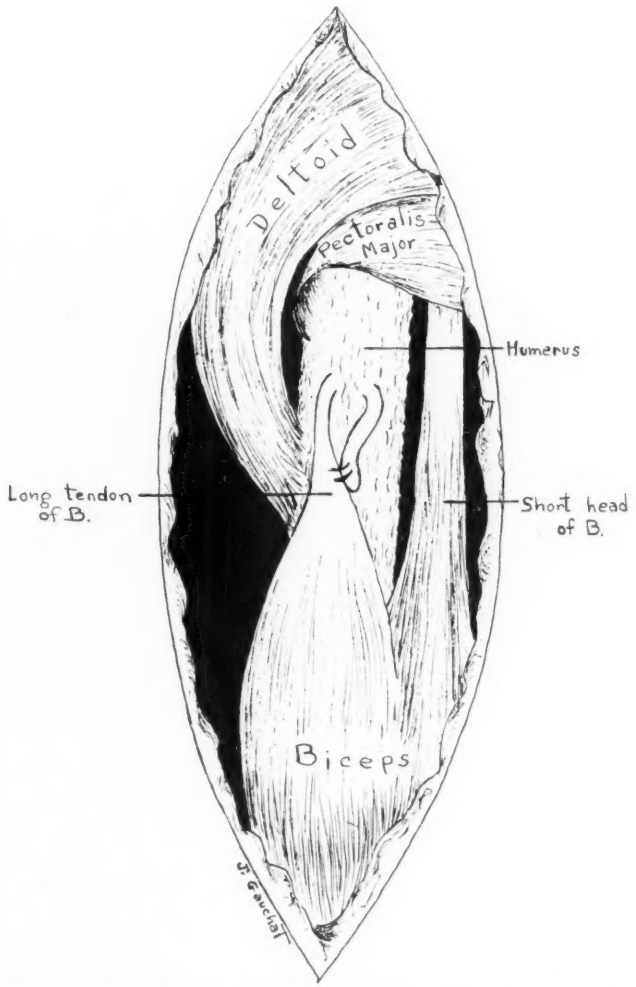

Fig. 6.—Diagrammatic drawing of the repair of the long tendon of the biceps brachii by suture to the shaft of the humerus (or to the bicipital groove).

Gilcreest was quite in accord with Keen's statement that "it seemed strange to him that surgeons so rarely operated on these patients when one sees so many cases of impaired function in those not operated on and such good results in those operated on early." Ludington was no less emphatic in stating that the proper treatment is operation. He stated that palliative measures offer no prospects of relief and that the disability is permanent unless corrected by operative means.

Although we are not quite willing to go as far as these authors have we are fully convinced that operation is indicated in the majority of cases, particularly in those patients who are to continue at laborious work and who have no co-existing disease that might be a contraindication to operation. This opinion is based on a personal experience of 50 cases, of which number there were performed 26 operations with very satisfactory results.

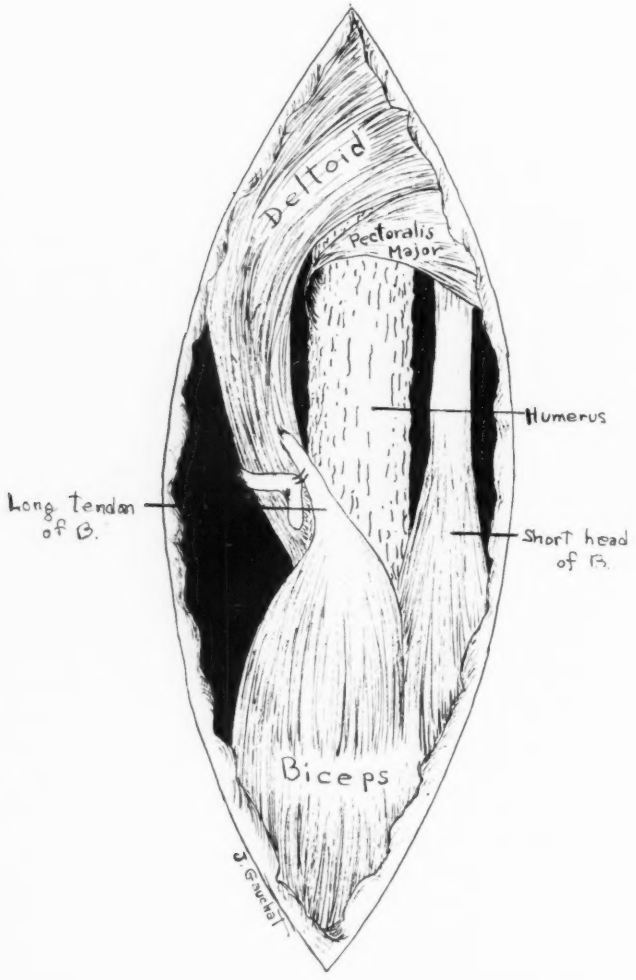

Fig. 7.—Diagrammatic drawing of the repair of the long tendon of the biceps brachii by suture to the insertion of the deltoid muscle.

For ruptures of the long tendon, one or a combination of several methods of repair may be chosen. These methods are as follows:

1. Suture of the long tendon to the short head (Bazy)
2. Suture of the long tendon to the coracoid process (Perthes)
3. Suture of the long tendon to the bicipital groove or to the humerus (Roloff)

4. Suture of the long tendon to the insertion of the deltoid (Cotton and Morrison)
5. Suture of the long tendon to the insertion of the pectoralis major (Hoffman)
6. Suture of the long tendon to the coracoid process, first passing the tendon through the region of the short head (Trethowan and Gilcreest).

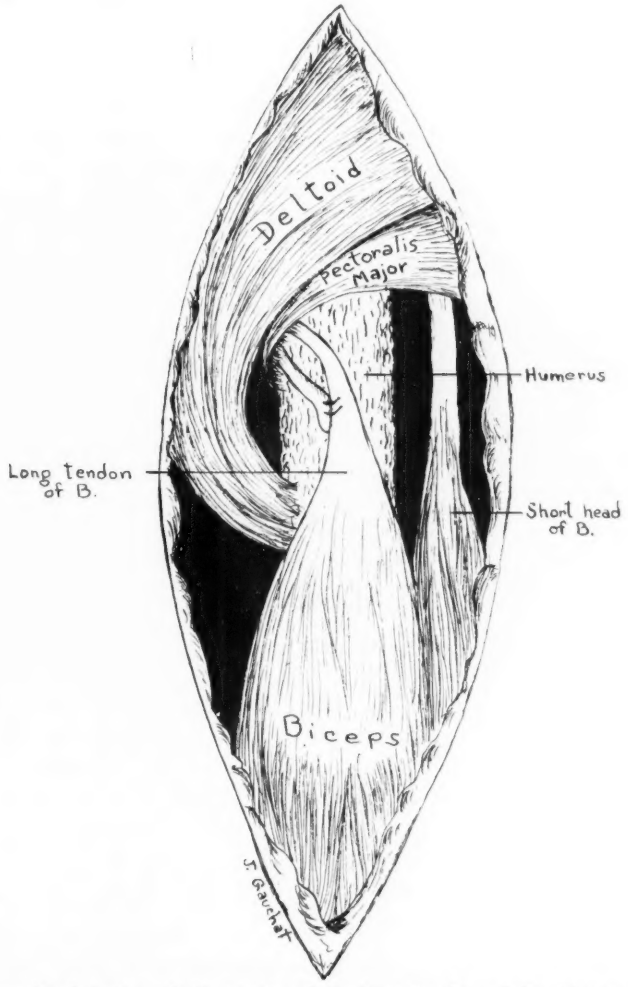

Fig. 8.—Diagrammatic drawing of the repair of the long tendon of the biceps brachii by suture to the insertion of the pectoralis major muscle.

In our previous communication we agreed with Gilcreest that if there is sufficient tendon it is better to combine the first two methods, that is, slipping the long tendon through and suturing it to a portion of the short head and also suturing the end of the tendon to the coracoid process. In one of our earlier cases, we were able, after weaving the long tendon through the short head, to fasten the end of the tendon through a hole drilled in the coracoid process, which gave not only a high but a very firm attachment.

This operation, however, is technically quite difficult and requires a fairly large exposure. After a larger experience, we now feel that the simpler operation of suturing the long tendon to the insertion fibers of the pectoralis major is much more practical. It is more easily done and the end results are most satisfactory. The fibrous expansion of the pectoralis major muscle with the transverse ligament forms the roof of the bicipital groove and, therefore, the

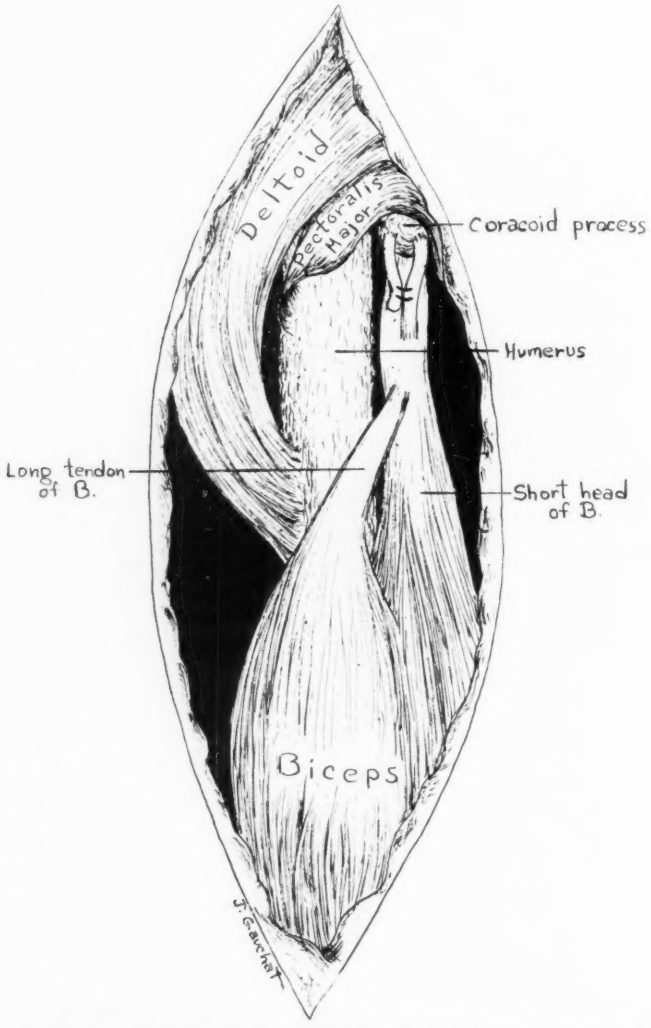

Fig. 9.—Diagrammatic drawing of the repair of the long tendon of the biceps brachii by suture to the coracoid process after passing the tendon through the short head of the biceps.

suturing of the long tendon to the fibrous expansion of the pectoralis major maintains the normal alignment of the muscle. On the other hand, suturing of the long tendon to the insertion of the delotid muscle results in a pull laterally, while suturing of the long tendon to the coracoid process or short head tends to produce a pull of the muscle medially. The suturing of the long tendon to the insertion fibers of the pectoralis major muscle also gives the long tendon a new

site of origin, higher than that with the fixation at the insertion of the deltoid muscle. The site of fixation to the expansion fibers of the pectoralis major muscle is essentially the same as the site of fixation of the distal portion of the divided long tendon as advised by Nicola in his operation for habitual dislocation of the shoulder, and which has been found in no way to impair the function of the biceps muscle. Also, suturing of the long tendon to the fibrous expansion of the pectoralis major muscle does not require as much long tendon as is necessary in suturing the long tendon to the coracoid process of the scapula.

For long tendon ruptures of the low type and for ruptures of either the upper or lower musculotendinous junctions it may be necessary to resort to fascia lata transplantation, or some other plastic procedure. With the site of rupture at the upper musculotendinous junction but with the long tendon fixed and competent we have sectioned the distal end of the long tendon and reimplanted the end in the muscle at a more distal site, thus elevating the muscle belly in an attempt to correct the muscle deformity and restore normal function of the muscle.

For ruptures of the lower tendon, several methods of repair have been suggested. Kirschner employed fixation of the tendon to the radius with a nail or, if this is not possible, he sutures the tendon to the neighboring soft parts. Leavitt scarified the periosteum and reattached the distal tendon by means of a hole drilled through the margin of the bicipital tuberosity. Bauman demonstrated a method of suturing the distal tendon by transfixing the tissues with sutures from the end of the tendon carried through to the posterior aspect of the forearm, where they are tied over gauze to hold the tendon in place.

Dobbie, in an analysis of 51 previously unreported cases of avulsion of the distal tendon, stated that there were many different methods of accomplishing repair of a ruptured distal tendon. In brief, these methods consisted of either a replacement of the avulsed distal tendon to the radius or an attachment of the distal tendon to structures in the forearm, other than the radius, that is, lacertus fibrosus, brachialis anticus muscle, or the ulna. Dobbie, in his own case, obtained a satisfactory result by splicing the distal tendon into the well-defined tendon of the brachialis anticus at its insertion into the base of the coronoid process of the ulna.

Fascial transplants, usually autogenous, have been used for fixation of the distal tendon by means of either loops around or drill holes in the shafts of the radius or ulna. Plummer, in his case, using ox fascia, approximated the distal tendon by means of drill holes through the ulna shaft adjacent to the bicipital tuberosity of the radius. Gallie used fascial grafts either looped around the shaft or through a drill hole in the bicipital tuberosity.

Fixation of the distal tendon to the radius or ulna, no doubt, gives a very firm reattachment but is a difficult operative procedure requiring a rather extensive dissection. Such operative procedures, therefore, are probably not worth while, since uniformly good results are obtained by the more simple operative procedures.

One might also comment on the added trauma to the deeper structures of the antecubital space, incidental to subperiosteal dissection or drilling of either

the radius or ulna in the more difficult or extensive operative procedures. An example of this sequel was observed in a patient referred to us from another city. The patient had previously suffered a rupture of the distal tendon of the biceps brachii and the method of repair was that of fixation of the distal tendon through a drill hole placed in the bicipital tuberosity of the radius. The immediate result was satisfactory but later there developed a hard lump or mass in the hollow of the elbow and upper forearm, which not only caused considerable discomfort but interfered with function, chiefly flexion of the elbow and supination and pronation of the forearm. The lump proved to be a large ossified mass contiguous with the radius, probably a periosteal osteoma or an ossification of a hematoma, that ultimately necessitated surgical excision. Such an occurrence of an ossified mass is somewhat analogous to the localized ossification or myositis ossificans that occasionally follows severe elbow injuries, particularly dislocations of the elbow.

In our previous communication, we stressed the importance of obtaining maximum relaxation and approximation of the torn muscle or tendon during the period of immobilization of the extremity. This optimum position for function was premised on the condition that if stiffness of the shoulder did occur, it would disable the patient as little as possible and would be advantageous to start from in securing a wide range of motion. This position, described also by MacGregor, was similar to the position of military salute, in which the arm is in moderate abduction and external rotation, the forearm in midposition and the elbow flexed to about 115 degrees.

However, after further experience we feel that it is more practical to immobilize the extremity immediately after operation in a sling with a reinforcement of light molded plaster of Paris or a Velpeau dressing for a period of ten to 14 days postoperatively, after which the extremity is placed in the so-called optimum position for function. The latter may be accomplished by using a special abduction arm splint if the patient is to remain ambulatory, or the patient may be put in bed for two to three weeks with the extremity maintained in the optimum position for function by means of balanced suspension with the overhead pulley. Convalescence is aided by the use of local heat, light massage, and increasing active exercises.

SUMMARY

1. The subject of ruptures of muscles and tendons, with particular reference to rupture of the biceps brachii, is reviewed and discussed together with a report of 50 cases of rupture of the biceps brachii personally observed during a fourteen-year period.

2. Ruptures of the biceps brachii often go unrecognized and untreated. This is due to the fact that many general practitioners and surgeons are not familiar with the condition. Mistaken diagnoses include conditions, such as strain or sprain of shoulder, rupture of supraspinatus tendon, subdeltoid bursitis, arthritis, periarthritis, neuritis, and rupture of blood vessels.

3. The increasing frequency of occurrence or rather diagnosis is due to the continued "biceps mindedness" in the diagnosis of shoulder conditions by our resident staff.

4. The 50 cases of rupture of biceps brachii include ruptures of the long tendon, ruptures at the musculotendinous junction, and ruptures (avulsion) of the distal tendon.

5. Twenty-nine of the 50 patients with rupture of the biceps brachii were subjected to operation. The results following operation were very satisfactory in all but one case.

6. The character of treatment depends upon the degree and site of the rupture and the age and occupation of the patient. However, in most instances operation is the method of choice. The various types of operation for ruptures of the biceps brachii are discussed in detail.

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MOTOR RESPONSES OF SPATIALLY TRANSPOSED INTESTINAL LOOPS

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THE exteriorized, skin-covered intestinal loop has offered a method of studying the motor behavior of the intestine under a variety of conditions without interrupting the normal functions of the loop or introducing factors of interference such as intraluminal foreign bodies.[1-5]

Alvarez[6, 7] has shown that the rates of rhythmic contraction both of surviving isolated loops and of loops in continuity in the saline bath are constant for a given intestinal loop and vary inversely with the distance from the pylorus. He has expressed the belief that this movement is myogenic in origin and does not depend on nerve cells. Krishnan,[8] Castleton,[9] and others have obtained similar results. Douglas and Mann[2] confirmed this property of the intestine using the exteriorized, skin-covered loop.

After a fast of forty-eight hours, during which time the animals receive only water, the exteriorized loop shows minimal activity. The introduction of a stomach tube in sham feeding or the administration of various liquids or solids causes a sudden burst of activity which is sustained in the case of a solid meal for hours. This motor response consists of a marked increase in tonus waves and tonus changes, irregular segmenting movements, and peristaltic waves which occur frequently but irregularly.[5] This feeding response is not mediated by the vagus nerves but it does depend on the continuity of the intestine for its mediation. Partial interruption of continuity by short-circuiting operations gave indefinite results, while complete isolation of the loop resulted in the absence of the feeding response.[3, 4] Douglas and Mann[3, 4] demonstrated that the time of the feeding response varies directly with the distance from the pylorus. In an animal having two loops at different distances from the pylorus, the response in the loop at the higher level always occurred before that in the lower loop. Interruption of the gut proximal to the loop and anastomosis in such a fashion as to prevent immediate union of muscular coats and enteric nerves did not affect the motor response.

In the present study it is proposed to investigate the behavior of an exteriorized intestinal loop in respect to these two functions, rate of rhythmic contractions and time of feeding response, both before and after transplantation to another site of the intestinal tract. All operations were performed with the animal under ether anesthesia, sterile technique being used. Trained dogs, fasted for forty-eight hours, were used; records were obtained by a tambour-Brodie bellows air-displacement recording system. The behavior of the loop was studied before and after the feeding of a meal of fresh raw horse meat. Fasting tracings

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of from three-fourths to one hour were obtained, and tracings were continued two hours after feeding. When the loop had been standardized, it was transplanted to another site in the intestinal tract. Thus, a loop of jejunum was transplanted to the ileum, or a loop of ileum was transplanted to a position in the jejunum. The segment transplanted was always anastomosed in isoperistaltic fashion. The incision for this secondary procedure was always placed on the side of the abdomen opposite that on which the exteriorized loop lay. The loop into which the exteriorized segment was to be intercalated was selected and its position in the intestinal tract was carefully noted. The anastomoses were all of the side-to-side type, except when the segments were too short, in which case end-to-end anastomoses were used. The weakest point in the technique is the fact that after the anastomoses are completed, three rents remain in the mesentery in such position that they cannot be closed by suture; thus, a predisposition to internal herniation and strangulation obtains. The incisions were healed and the animals were ready for study after two or three weeks. In two animals the effects of simple sectioning and anastomosis of the proximal and distal ends of the exteriorized loop were studied. Studies were made after complete recovery from operation, six months later, eighteen months later, and two years later.

RESULTS

The most common type of activity seen was the irregular segmentation. This appeared to be a summation of simpler movements with phase differences. Peristaltic waves were very uncommon in the fasting loops but were regularly seen after feeding. They involved the entire loop in the contraction, and by light finger pressure the passage of contents in an aboral direction was perceptible. The kymographic tracings of such activity show that these waves have the form of the P or, more commonly, the K waves described by Krueger, Lampe, and Reid.[10] Interesting as the analysis of the type of wave form is, the real criterion which we used for the purpose is free of all objections that might be attributed to the recording method. We were interested in the motor feeding reaction, a response characterized by a marked increase in the tonus waves and tonus changes, irregular segmentation, and peristaltic waves.

In all experiments the number of rhythmic contractions per minute and the time of feeding response were noted. The loops were standardized in this fashion before being transplanted. After recovery from transplantation, usually a period of from two to three weeks, these two sets of data were again determined. These results are given in Table I.

It was found that the number of rhythmic contractions per minute of any individual loop remained remarkably uniform throughout. The rates of those loops in the jejunum and close to the ligament of Treitz were around 17 to 18 per minute; the rate decreased uniformly until it was 11 to 12 per minute in those loops just above the ileocecal valve. The interval from feeding to the time of motor response in the loop (feeding reaction time) varied directly as the distance from the pylorus. It varied from one to five minutes in the loops of the upper part of the jejunum and seven to ten minutes or longer in the loops of the lower part of the ileum.

TABLE I. RHYTHMIC CONTRACTIONS PER MINUTE AND TIME OF FEEDING RESPONSE BEFORE AND AFTER TRANSPLANTATION OF INTESTINAL LOOP

	BEFORE TRANSPLANTATION				AFTER TRANSPLANTATION				
		RHYTHMIC CONTRACTIONS PER MINUTE		FEEDING RESPONSE (MIN.)	SITE TO WHICH LOOP WAS TRANSPLANTED	RHYTHMIC CONTRACTIONS PER MINUTE		FEEDING RESPONSE (MIN.)	NUMBER OF EXPERIMENTS
ANIMAL	SITE OF LOOP	AVERAGE	RANGE			AVERAGE	RANGE		
1	18 cm. below ligament of Treitz	17	16-18	1	30 cm. above ileocecal valve	15	14-16	6-20	18
2	15 cm. below ligament of Treitz	17	17-18	1	20 cm. above ileocecal valve	15	14 15	10-15	28
3	42 cm. below ligament of Treitz	17	17-18	1-6	10 cm. above ileocecal valve	14	13-14	10-15	25
4	48 cm. below ligament of Treitz	17	16-18	1-3	20 cm. above ileocecal valve	15	14-16	8-10	17
5	72 cm. below ligament of Treitz	15	14-16	5-10	20 cm. below ligament of Treitz	13	13-14	1-4	24
6	36 cm. above ileocecal valve	13	12-14	8-15	36 cm. below ligament of Treitz	12	11-13	1	15
7	18 cm. above ileocecal valve	13	12-13	7-18	40 cm. below ligament of Treitz	11	11-12	1-4	30
8	14 cm. above ileocecal valve	12	11-13	10-15	Proximal end of loop transected and anastomosed	11	11-12	10-15	18
9	15 cm. below ligament of Treitz	17	17-18	1-3	Both ends of loop transected and anastomosed side to side	15	14-15	1-4	23

After transplantation of the loop the rate of rhythmic contractions was seen to decrease by one or two contractions per minute regardless of whether or not the loop was transplanted higher in the tract than originally. For example, in Animal 5, which initially had a loop the mid-portion of which was 72 cm. below the ligament of Treitz, the original rate was 15 per minute. After the loop had been transplanted to a position higher in the jejunum the rate dropped to 13 per minute. In another loop (Animal 7) the rate dropped on transplantation to a higher level from an average of 13 per minute to an average of 11 per minute. The same condition was noted on transplantation of a loop from a higher to a lower position. For example, in animal 1 the original site of the loop was 18 cm. below the ligament of Treitz, where the average rate of rhythmic contraction was 17 per minute. When the loop was transplanted to a position low in the ileum the rate dropped to an average of 15 per minute.

The feeding reaction time varied directly with the distance from the pylorus. Loops which were originally high in the jejunum and had immediate

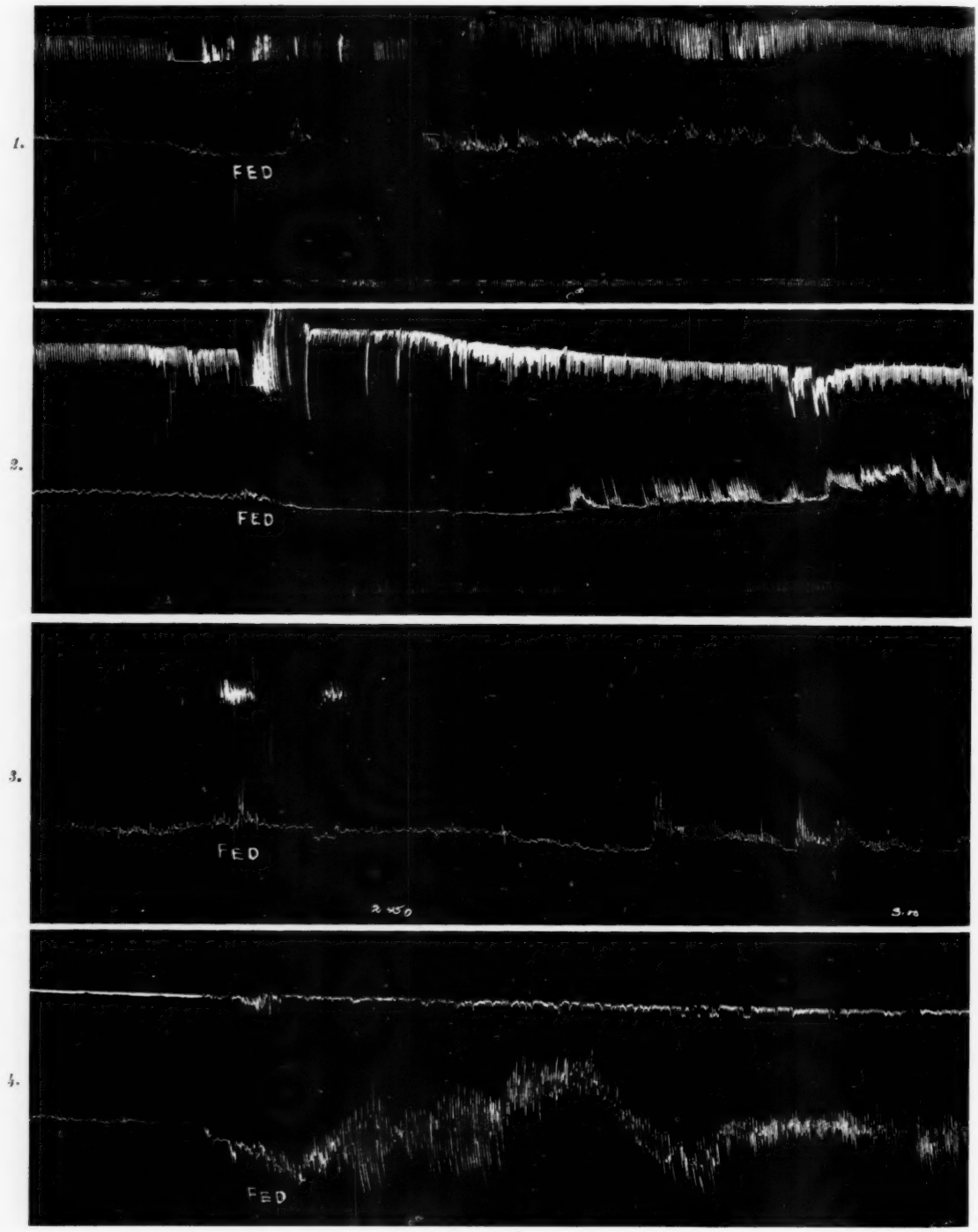

Fig. 1.—Kymograph records from top to bottom:

1. Feeding reaction in high jejunal loop (Animal 2).

2. Feeding reaction in high jejunal loop after transplantation to lower part of ileum (Animal 2).

3. Feeding reaction in low ileal loop (Animal 7).

4. Feeding reaction in low ileal loop after transplantation to higher part of jejunum (Animal 7).

Top line of each record is respiratory tracing; middle line is record of motor activity of loop; lower time tracing is in intervals of five seconds.

feeding reaction time (one to three minutes), on being transplanted to a position lower in the ileum had feeding reaction times of eight to twelve minutes; conversely, loops originally low in the ileum and having feeding reaction times of eight to twelve minutes or longer, on being transplanted high into the jejunum manifested immediate feeding reaction times (Fig. 1).

COMMENT

The fact that the time of the motor response to feeding in a given loop varies directly with the distance of that loop from the pylorus bears out the original hypothesis of Douglas and Mann[3] that some form of motor activity passes down the length of the small intestine after feeding. It would appear from such observation that the response does not depend on the integrity of the mesenteric nerves. The behavior of the rhythmic contractions presents some unusual features. The fact that the rate is decreased when a loop is moved would seem to indicate that the rate of rhythmic contraction is a property inherent in the loop itself and does not depend on the continuity of the intestine. Alvarez,[6, 7] Douglas and Mann,[2] and others have demonstrated that the rate of rhythmic contraction is consistent for any given loop with a variation of ± 1 contraction per minute. The rate was found by them to be a function of the distance from the pylorus, the loop at the highest level having the highest rate. This rate was not affected by degenerative section of vagal or splanchnic nerves.

We attempted to control this point by sectioning the afferent and efferent limbs of the exteriorized loops and anastomosing them without the added feature of transplantation. In one dog with a low loop there was a decrease from an average of 12 to 11 per minute. However, only the afferent limb of this ileac loop lent itself to sectioning and only one anastomosis was made. In a dog with a high jejunal loop both limbs of the loop were sectioned and anastomosed. Here the rate fell from 17 to 15 per minute.

It is interesting to compare a finding of Castleton[9] from his study of electrographic tracings of intestinal loops; he found that there was a decrease in rate of rhythmic contraction after denervation. Later he isolated completely an exteriorized loop which he had studied previously. Whereas before isolation the rate of rhythmic contraction had been 17 or 18 per minute, after isolation the rate decreased to 13 or 14 per minute.

We have no explanation for our observation except to compare it with the drop in rate seen after complete isolation. Further studies with end-to-end anastomoses would perhaps lead to an explanation. Cannon and Murphy[11] have shown, for example, that the efficiency of the gut after end-to-end suture is not compromised. However, after side-to-side anastomoses there is some disturbance of function, because food tends to accumulate in the blind ends formed by the opposed loops.

Douglas,[12] in 1948, working independently in Edinburgh on dogs, found a fall in frequency of the order of 20 per cent when a jejunal loop is transplanted or isolated. He noted a similar fall in frequency following section and reanastomosis of the intestine proximal to a jejunal loop, whereas he found no change in frequency following distal section.

SUMMARY

Exteriorized skin-covered loops of jejunum and ileum were studied in respect to their rhythmic contractions and their motor responses after feeding. These rhythmic contractions were found to be constant for any given loop of intestine with a variation of ±1 contraction per minute. The motor feeding response was found to occur more rapidly in those loops which were nearer the pylorus than in more distal loops.

The positions of the intestinal loops were changed with respect to their distance from the pylorus. Loops which were originally high in the jejunum were transplanted without interference with their blood and nerve supply to positions in the lower part of the ileum. Likewise, loops that were low in the ileum were transplanted to a position high in the jejunum. These operations of intercalary transplantation of intestinal loops have been successfully carried out in dogs although they involve several inherent risks. Several preparations, however, are in good condition four years after operation.

The rhythmic contractions in the transplanted loops were of the same order as when the loop was in its original position minus one or two contractions per minute. The rate of rhythmic contractions is thus seen to be a property of the loop itself; it does not depend on the relative position of the loop in the intestinal tract.

The time of the feeding response depends on the relative position of the loop in relation to the remainder of the intestinal tract. The more orad the loop, the quicker does the motor response occur after feeding. These findings support the hypothesis that after the ingestion of food there is a wave of activity passing down the length of the small intestine.

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THE MECHANISM OF SHOCK IN PERITONITIS

A Study of the Hemodynamics of Shock Occurring in Peritonitis Experimentally Produced in Dogs

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IN A study of traumatic shock associated with battle wounds,[1] it was noted that severe and irreversible shock frequently occurred with penetrating abdominal wounds in which there was fecal contamination of the peritoneal cavity. Certain patients in shock from abdominal wounds had a good initial response to therapy and then developed a secondary fall in arterial pressure which did not respond to further transfusions even though the blood volume was restored to normal. Richards[2] noted that the decrease in blood volume was less marked in the shock of abdominal injury than in the shock of skeletal trauma and suggested that peritonitis and infection may play an important role in the production of shock following abdominal wounds.

In order to elucidate the hemodynamics of the shock accompanying peritonitis, a study was made of the experimental shock produced by fecal contamination of the peritoneal cavity of dogs.

METHOD

The studies were carried out on mongrel dogs. Plasma volume was determined by a dye method.[3] The oxygen consumption was measured directly using a Benedict-Roth closed circuit spirometer. The cardiac output was determined by the Fick principle.[4] A catheter was introduced through the right external jugular vein and passed into the right ventricle under fluoroscopic vision.[5] Mixed venous blood from the right ventricle was obtained through the catheter. The arterial pressure was measured with a mercury manometer by direct puncture of the femoral artery. The total peripheral resistance (TPR) in dynes per second per centimeter^{-5} was calculated from the formula[6]:

$$\text{T.P.R.} = \frac{\text{Arterial Pressure (mean) mm. Hg.} \times 1332}{\text{Cardiac Output (Ml./sec.)}}$$

After completing the control studies, the dog was anesthetized with sodium pentothal and a small midline abdominal incision was made. Approximately 50 c.c. of a thin suspension of feces in saline solution were introduced into the peritoneal cavity. The dog was then allowed to recover from the effect of the anesthetic agent. One group of dogs was allowed to progress into shock without therapy. In a second group of dogs an attempt was made to maintain the blood volume at the normal level by plasma and blood transfusions. Because of the rapid loss of plasma into the peritoneal cavity it was impossible to maintain a completely normal plasma volume at all times, but it was possible to maintain

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the arterial pressure until a terminal fall. When the fall in arterial pressure occurred the studies were repeated. The rectal temperatures of the animals were followed during the course of the experiments and ranged between 101 and 104° F.

Autopsies were performed on all animals, at which time the blood from the heart was cultured.

RESULTS

The group of seven dogs which was untreated developed severe shock in three to ten hours. The shock was characterized by a decrease in arterial pressure, hemoconcentration, low plasma volume, and a fall in cardiac output (Table I). The average reduction in plasma volume in the five dogs in which it was

TABLE I

WT. (KG.)	HEMATOCRIT READING	PLASMA VOLUME (C.C.)	CARDIAC OUTPUT (L. PER MIN.)	MEAN ARTERIAL PRESSURE (MM. HG.)	TOTAL PERIPHERAL RESISTANCE	COMMENT
			Untreated Controls			
16.8	44.2	--	3.22	168	4170	Control
	59.2	--	0.77	50	5190	7′7″ after introduction of feces
16.7	42.7	--	1.68	114	5420	Control
	60.0	--	1.50	68	3620	10′5″ after introduction of feces
12.9	51.8	720	2.55	138	4320	Control
	65.7	440	1.67	96	4590	9′30″ after introduction of feces
16	45.6	1080	2.60	110	3380	Control
	71.3	610	0.83	60	5770	8′50″ after introduction of feces
15	48.2	1000	2.00	150	5990	Control
	65.2	520	1.20	72	4790	6′42″ after introduction of feces
12.2	50.1	640	2.20	138	5010	Control
	61	420	0.49	50	8150	3′ after introduction of feces
15.9	46.9	840	1.80	155	6880	Control
	72.3	350	0.40	40	7990	10′13″ after introduction of feces
			Inadequately Treated Shock			
12.3	51.5	650	1.45	150	8270	Control
	62.0	480	0.62	58	7470	10′45″ after introduction of feces, 1300 c.c. plasma
12.0	42.4	800	1.82	130	5710	Control
	58.5	520	0.92	70	6080	8′37″ after introduction of feces, 1150 c.c. plasma
18.6	49.0	--	3.27	120	2930	Control
	70.2	--	2.00	75	3000	9′5″ after introduction of feces, 1000 c.c. plasma, 400 c.c. blood
			Adequately Treated Shock			
14.1	46.2	780	1.09	125	9160	Control
	53.2	670	1.78	60	2690	5′45″ after introduction of feces, 925 c.c. plasma
15.9	53.1	790	2.00	118	4710	Control
	54.8	780	2.20	68	2470	9′15″ after introduction of feces, 1950 c.c. plasma
12.3	52.2	630	3.36	105	2500	Control
	55.4	590	3.15	70	1780	11′0″ after introduction of feces, 1300 c.c. plasma
15.4	50.2	790	2.22	136	4900	Control
	50.0	810	2.18	76	2790	10′25″ after introduction of feces, 1350 c.c. plasma
13.6	58.4	630	2.18	168	6160	Control
	51.9	680	2.83	62	1750	8′30″ after introduction of feces, 1225 c.c. plasma

measured was 44 per cent. The change in total peripheral resistance was variable, there being an increase in five instances and a decrease in two instances. The average total peripheral resistance for the group was 5,020 during the control period and 5,730 during the period of shock. After death 350 to 550 c.c. of fluid with an average protein content of 2.5 Gm. were found in the peritoneal cavity.

In a second group of three dogs, the shock was inadequately treated as shown by reduced plasma volume or marked hemoconcentration. This group showed essentially the same findings as the untreated group.

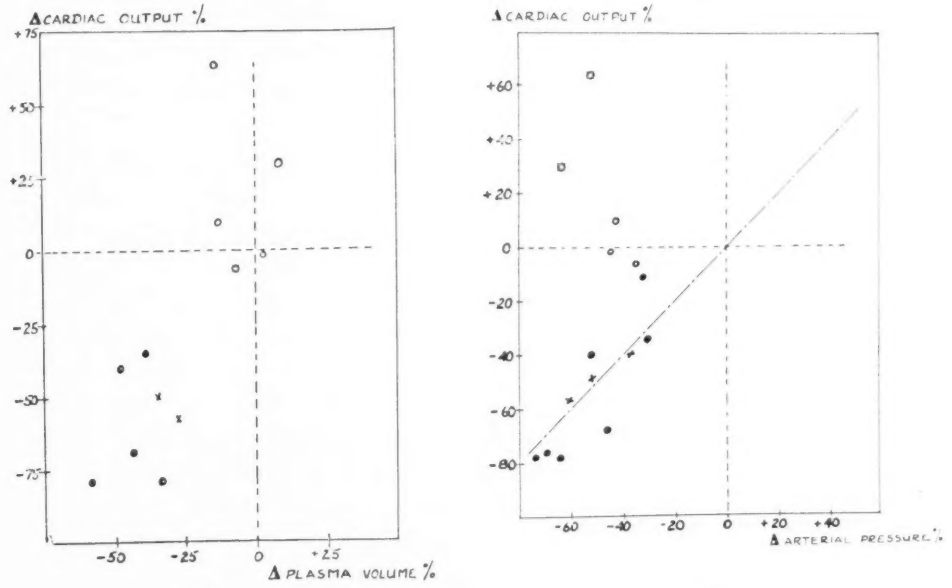

Fig. 1.—Graphic comparison of the change in cardiac output with change in plasma volume (*A*) and arterial pressure (*B*) following production of shock by peritonitis. Solid circles represent untreated dogs, open circles represent adequately treated dogs, and crosses represent inadequately treated dogs. If the peripheral resistance remained constant with changes in cardiac output and arterial pressure the values would fall on the diagonal line in *B*.

In the group of five animals adequately treated, as defined by a relatively normal blood volume, a marked fall in arterial pressure occurred in six to eleven hours. The animals appeared extremely ill and were usually semicomatose. The plasma volume was normal or only slightly decreased and there was no evidence of hemoconcentration. The mean arterial pressure was markedly reduced but the cardiac output remained unchanged or was somewhat elevated above the control level (Table I and Fig. 1). As a consequence, the total peripheral resistance was reduced in all instances, the average value for the group being 5,490 during the control period and 2,300 during the period of shock. Culture of the blood consistently yielded bacilli of the colon group. It is of interest that transfusion therapy did not prolong life.

DISCUSSION

The shock produced by peritonitis in the untreated animals resembles shock due to hemorrhage,[2, 7, 8] in that the arterial pressure and cardiac output are re-

duced and the peripheral resistance is maintained. The reduction in cardiac output can be attributed to a considerable reduction in plasma volume, which is caused by the loss of fluid into the peritoneal cavity. Mention should be made of the role of hemoconcentration in the shock of peritonitis. Seligman, Frank, and Fine[9] have emphasized that hemoconcentration increases peripheral resistance, because of the increased viscosity of the blood. In the untreated animals the hematocrit reading is markedly increased, and the consequent increased viscosity of the blood must play a role in maintaining the peripheral resistance.

If the plasma volume is sustained by infusion of plasma, hypotension eventually develops even though the blood volume is normal and the cardiac output is not reduced. This hypotension is attributed to a loss of peripheral resistance due to vasodilatation. In the animals adequately treated with plasma the hematocrit reading is held at normal levels and hence increased viscosity of the blood is lacking.

The fall in arterial pressure which occurs in the adequately treated shock of peritonitis must be differentiated from the irreversible shock which may follow severe hemorrhage. It has been shown that if the arterial pressure of a dog is kept below 40 mm. Hg for one hour by repeated hemorrhages, reinfusion of the blood will not restore the pressure, and death eventually ensues.[10] The cardiac output is usually low although there may be a transient increase immediately after transfusion.[11] In the present experiment a prolonged period of hypotension was avoided, and the cardiac output remained normal or was slightly increased. Hence, the mechanism of the fall in the arterial pressure in the adequately treated shock of peritonitis is believed to be different from that which occurs in the irreversible shock of hemorrhage.

The reason for the vasodilatation and consequent decrease in peripheral resistance in the adequately treated group of dogs is not known. The tremendous vasodilatation in the splanchnic area, due to the peritonitis, must be a factor of major importance. Blalock[12] observed a fall in blood pressure without fall in cardiac output after the introduction of bile, pancreatic or gastric secretions into the peritoneal cavity of dogs. The fall in blood pressure was attributed to dilatation of the small intraperitoneal vessels. The importance of the infection itself is less easy to evaluate. Shock in animals consequent to infection with bacteria of the Clostridium group has been described.[13, 14] This shock was not caused by a lowered blood volume although the cardiac output was reduced and the peripheral resistance was increased. The role of the vasodepressor principle (VDM) described by Shorr and associates[15] in hemorrhagic and tourniquet shock cannot be evaluated from the present studies.

In recent years there has been a proper emphasis on the importance of the diminished blood volume in the pathogenesis of traumatic shock. Such emphasis has tended to obscure the importance of the peripheral vascular bed. The clinical studies of Merrill and co-workers[16] have shown that vasodilatation is of importance in the shock associated with stab wounds of the chest as evidenced by the marked fall in arterial pressure in the presence of a relatively normal cardiac output and blood volume. The present study suggests that both the

diminution in blood volume and the reaction of the peripheral vascular bed are important in the pathogenesis of the shock of peritonitis.

SUMMARY

1. Shock secondary to peritonitis was experimentally produced in dogs by introducing feces into the peritoneal cavity.

2. In untreated animals the shock appeared to be due to diminution of plasma volume as a result of loss of fluid into the peritoneal cavity with consequent reduction in cardiac output and fall in arterial pressure.

3. In animals treated with plasma there was a fall in blood pressure in the presence of a normal blood volume and cardiac output. The hypotension was a result of a loss of resistance in the peripheral vascular bed.

4. This study suggests that both the loss of plasma into the peritoneal cavity and vasodilatation particularly in the splanchnic area are implicated in the pathogenesis of the shock of peritonitis. Under these circumstances, transfusion therapy is ultimately ineffectual.

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ACUTE PERFORATIONS OF STOMACH AND SMALL BOWEL ULCERATIONS

An Analysis of Three Hundred Sixty-Two Consecutive Cases

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IN THE ten-year period between 1938 and 1948, there have been 362 proved cases of acute, free perforation of gastrointestinal ulceration at the St. Louis City Hospital. Of these, 318 patients were treated surgically and the remaining 44 cases were proved at autopsy. Those few patients who were treated conservatively, either because of indecision in diagnosis or because of the patient's refusal of surgery, and who did not come to autopsy, have not been included. A summary of these cases is being presented to review the established facts and their relationship to one another, as well as to present a few less publicized aspects of the disease.

Incidence.—During this ten-year period there were 170,236 hospital admissions, and 0.21 per cent of these were for acute intestinal perforations.

Years.—The number of operations for perforations has varied with each year, the yearly average being 31. Since 1938, the operative mortality has gradually decreased, which possibly can be explained by the introduction of the sulfonamide drugs and penicillin.

Seasons.—The usual, and unexplained, increased occurrence of perforation in spring was noted. The increased operative mortality in winter is probably explained by the high incidence of respiratory infection, by the patient's decreased resistance to infections, and perhaps by a delay in his entering the hospital because of the weather.

Occupation.—Of these patients, 30.3 per cent were either unemployed or retired. Twelve truck drivers comprised the most frequent occupation; there were 10 each of salesmen and clerks, and bartenders ranked third with 7. Sixty per cent were either married or widowed; 27 per cent were single and 12 per cent were divorced or separated.

Sex.—Seventeen patients (4.7 per cent) included in this series were women, and all 9 who were treated surgically lived. Apparently because the diagnosis was not considered so frequently for women, the diagnosis was not made in the remainder until autopsy, for a total mortality of 47.1 per cent.

Ages.—Two-thirds of all the patients were between 30 and 60 years of age, the 40 year age group having been the most common. The mortality gradually rose from 0 per cent in the teens to 73.7 per cent for the 70 year age group. There is no doubt that the mortality of those in the 80 year age group would have been higher had there been many more of them. The total postoperative mortality was 18.2 per cent.

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TABLE I. YEARLY VARIATIONS OF INCIDENCE AND MORTALITY

YEAR	OPERATIVE CASES	DEATHS	PER CENT MORTALITY
1938	24	10	41.7
1939	37	11	29.7
1940	28	5	17.9
1941	38	5	13.2
1942	37	7	18.9
1943	31	3	9.7
1944	25	4	16.0
1945	40	8	20.0
1946	33	4	12.1
1947	24	1	4.2
1948	1		
Total	318	58	18.2

TABLE II. SEASONAL VARIATIONS OF INCIDENCE AND MORTALITY

SEASONS	OPERATIVE CASES	PER CENT	DEATHS	PER CENT MORTALITY
Winter	63	19.8	18	28.6
Spring	92	28.9	11	12.0
Summer	83	26.1	12	14.5
Autumn	80	25.2	17	21.3
Total	318	100	58	18.2

TABLE III. VARIATIONS OF AGE AND MORTALITY

AGES IN YEARS	OPERATIVE CASES	POST-OPERATIVE DEATHS	PER CENT OPERATIVE MORTALITY	TOTAL CASES	PER CENT	TOTAL DEATHS	PER CENT MORTALITY
0 to 9	0	0	0.0	1	0.3	1	100.0
10 to 19	4	0	0.0	4	1.1	0	0.0
20 to 29	48	3	6.3	48	13.3	3	6.3
30 to 39	71	6	8.5	73	20.2	8	10.9
40 to 49	76	13	17.1	84	23.2	21	25.0
50 to 59	70	15	21.4	81	22.4	26	32.1
60 to 69	34	12	35.3	49	13.5	27	55.1
70 to 79	14	9	64.3	19	5.2	14	73.7
80 to 89	1	0	0.0	3	0.8	2	66.7
Total	318	58	18.2	362	100	102	28.2

Past History.—It is usually difficult to obtain a good history from a patient so acutely ill as these, and perseverance does not always win. A patient who has a second perforation often makes his own diagnosis. Fifteen patients (4.1 per cent) were encountered who had had previous perforations; one had had two. Fifty-one patients apparently had been in good health with no symptoms and no knowledge of having a peptic ulcer until perforation. Histories of symptoms compatible with peptic ulcer were obtained from 228 patients, and in an additional 70 the symptoms were referable to the upper gastrointestinal tract, but were rather atypical for peptic ulcer.

The duration of symptoms varied widely from several days to thirty-seven years. Approximately one-third gave histories of less than one year's duration. Another third dated their symptoms for one to five years, and the remaining third had symptoms for longer than five years.

A history of bleeding in the form of hematemesis or melena was obtained from 89 patients, the latter having been more common. Hematemesis occurred with the perforation in 21 patients and of these, 7 were massive. Only 2 of the latter recovered.

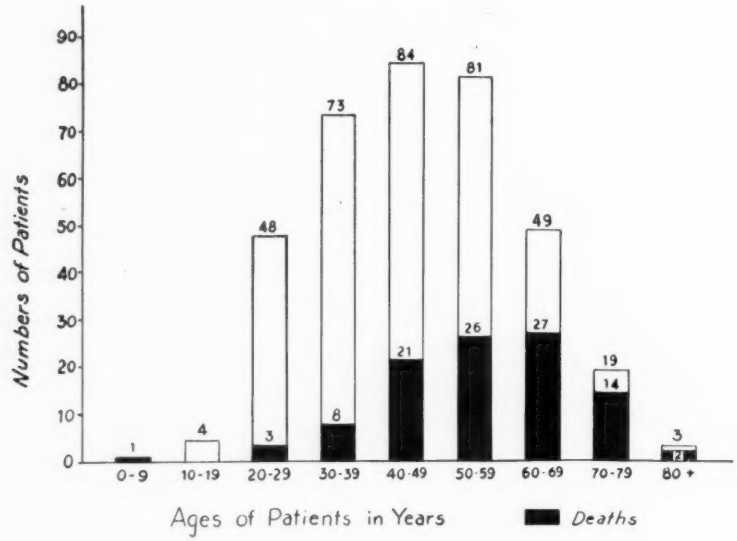

Fig. 1.—Ages and mortality.

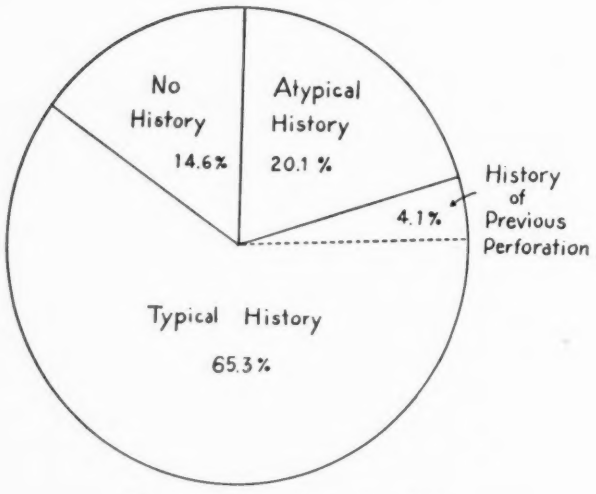

Fig. 2.—History of peptic ulcer.

Onset of Present Illness.—The patient's activity at the time of the perforation was about equally divided between rest and some kind of exertion. There were 65 who were actually lying down at rest or asleep and 27 were known to have been engaged in some activity involving abdominal strain such as carrying a heavy load or straining at stool. In contradistinction to those in whom the intra-abdominal pressure was raised by abdominal muscle

contraction, the pressure was directly increased by increasing the intraluminal pressure in those patients whose perforations followed meals or the drinking of carbonated beverages. It was noted that 46 patients had been drinking beer soon or immediately before the perforation. Other alcoholic and carbonated beverages preceded 12 more; 5 perforations followed a barium meal within twelve hours. Exclusive of drinking, it was reasonably well stated

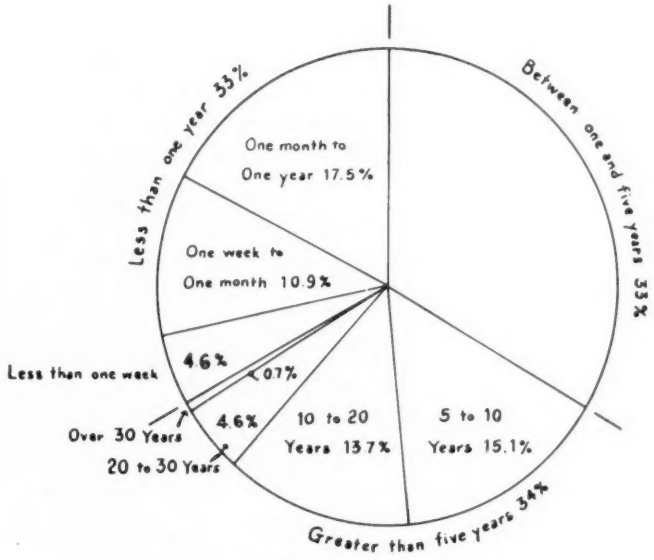

Fig. 3.—Duration of symptoms.

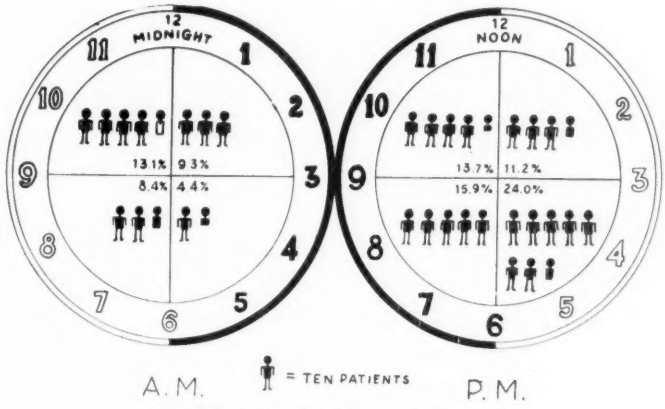

Fig. 4.—Time of day of perforation.

that 60 perforations occurred on an empty stomach and 45 soon after a meal. When the base of an actively penetrating ulcer becomes separated from the peritoneal cavity by merely a thin layer of peritoneum or fibrous tissue, not much change in pressure, motility, or acidity should be necessary to cause the complete break-through.

Late in the afternoon, the time of day soon before dinner and several hours after lunch, seemed to be the most common time for perforations to

TABLE IV. VARIATIONS OF HISTORIES

HISTORY	NUMBER OF PATIENTS	PER CENT
Typical	228	65.3
Atypical	70	20.1
No history	51	14.6
Total known histories	349	100

TABLE V. DURATION OF SYMPTOMS OF PEPTIC ULCER

DURATION OF HISTORY	NUMBER OF PATIENTS	PER CENT
Less than 1 week	13	4.6
1 week to 1 month	31	10.9
1 month to 1 year	50	17.5
1 year to 5 years	94	33.0
5 to 10 years	43	15.1
10 to 20 years	39	13.7
20 to 30 years	13	4.6
30 to 40 years	2	0.7
Total known durations	285	100

TABLE VI. History of Bleeding

HISTORY	NUMBER OF PATIENTS	PER CENT
History of melena	61	16.9
History of hematemesis	46	12.7
Hematemesis with perforation	21	5.8
Massive hematemesis with perforation	7	1.9
History of bleeding	89	24.6

TABLE VII. ACTIVITY AT TIME OF PERFORATION

ACTIVITY	NUMBER OF PATIENTS	PER CENT
Lying in bed	65	30.4
Idle or sitting in a chair	35	16.4
Mild activity	87	40.6
Activity with definite strain	27	12.6
Total known activities	214	100
Following alcoholic or carbonated beverages	58	16.0
Following barium meal	5	1.4

TABLE VIII. TIME OF DAY OF PERFORATION

TIME	NUMBER OF PATIENTS	PER CENT
12 Midnight to 3 A.M.	30	9.3
3. A.M. to 6 A.M.	14	4.4
6 A.M. to 9 A.M.	27	8.4
9 A.M. to 12 noon	42	13.1
12 noon to 3 P.M.	36	11.2
3 P.M. to 6 P.M.	77	24.0
6 P.M. to 9 P.M.	51	15.9
9 P.M. to 12 midnight	44	13.7
Total known	321	100

occur. The most uncommon time was 5 A.M., a time when most people (including alcoholics) are at rest, and usually the time of day when free gastric acidity is at its lowest ebb.

The majority of the patients gave a typical history of sudden onset of severe epigastric pain. The pain was epigastric or generalized in 329 (90.9 per cent), and among these the right lower quadrant was specifically included in 15 instances. The right upper quadrant was predominant in 24 and the left upper quadrant, in 2. The pain was apparently solely in the hypogastrium in 25 patients (6.9 per cent), and of these was mostly in the right lower quadrant 19 times, the left lower quadrant once. Eight patients (2.2 per cent), who were either senile or already suffering from an advanced disease, died without ever complaining of pain.

Referral of pain to one or both shoulders from diaphragmatic irritation by gastric contents was found in 150 patients (65.8 per cent), and was stated to be absent in 78. Referral of pain to the penis, apparently from irritation of serosa overlying the ureter and urinary bladder, was present in 7 (1.9 per cent).

Nausea and vomiting followed the onset of pain in 215 patients (63.2 per cent), and was stated to be absent in 125.

Because of the severity of the pain, most patients presented themselves early in the disease (about one-half within six hours), and fortunately so, because the mortality climbed rapidly with the number of hours before admission, thereby delaying surgical treatment.

TABLE IX. NUMBER OF HOURS OF DURATION OF PERFORATION BEFORE ADMISSION AND MORTALITY

NUMBER OF HOURS BEFORE ADMISSION	OPERATIVE CASES	POST-OPERATIVE DEATHS	PER CENT OPERATIVE MORTALITY	TOTAL CASES	PER CENT	DEATHS	PER CENT MORTALITY
0 to 6	204	19	9.3	207	57.2	22	10.6
6 to 12	66	16	24.2	67	18.5	17	25.4
12 to 24	22	10	45.4	25	6.9	13	52.0
Over 24	13	6	46.1	29	8.0	22	75.9
Undetermined	6			12	3.3	6	50.0
Hospital patient	7	7	100.0	22	6.1	22	100.0
Total	318	58	18.2	362	100	102	28.2

It is unfortunate, but interesting, to note that 22 perforations occurred in persons while they were patients in the hospital, and that all 22 patients died. Seven were recognized as surgical emergencies, but closures were attempted too late. A few others were recognized, but the patients' physical conditions were then too poor to permit operation. The remainder of the perforations were unsuspected and were discovered at autopsy. Several, however, could not have been diagnosed because they apparently had been terminal events of other diseases.

Examination.—The temperature, pulse, respiration, blood pressure, and white blood cell counts were recorded shortly after admission to the hospital in nearly all instances. The temperatures were relatively normal during the

TABLE X. VARIATIONS OF LEUCOCYTOSIS AND MORTALITY

W.B.C.	NUMBER OF PATIENTS	PER CENT	DEATHS	PER CENT MORTALITY
Less than 5,000	4	1.2	3	75.0
5,000 to 10,000	58	17.0	19	32.8
10,000 to 15,000	115	33.7	32	27.8
15,000 to 20,000	99	29.0	22	22.2
Greater than 20,000	65	19.1	8	12.3
Total white counts	341	100		

first six hours after perforation, only about 21 per cent having been definitely subnormal. They were found to climb gradually with the hours as bacterial peritonitis developed. More than one-half of the temperatures recorded after twenty-four hours were over 100° F. Some of those patients with a nearly normal temperature after twenty-four hours could possibly have had spontaneous sealing and have been escaping infection, but in others it represented a terminal fall.

The pulse and respiratory rates also began normally and tended to rise with the temperature and duration of perforation. After twenty-four hours, greater than one-half the pulse rates were over 100, and respiratory rates, over 30.

Blood pressures apparently varied with the individual. As has been pointed out numerous times, the patient appears to be in a shocklike state with prostration, and pale, cold, clammy skin, but without a lowered blood pressure. Only 11 patients (3 per cent) entered with systolic pressures below 80, mostly when after twenty-four hours, and the mortality of these was 72.7 per cent.

The white blood cell counts were moderately elevated between 10,000 and 20,000 in most cases. It was noted that the higher the count, the better the prognosis.

Thus, after twenty-four hours, the more ominous signs included an elevated or subnormal temperature, a rapid respiration, a low blood pressure, or low white count.

Diastase.—Determinations of blood diastase activity, to eliminate the possibility of acute pancreatitis, were obtained in about one-third of all cases and were found to be normal in 97.5 per cent of these. The remainder were moderately elevated between 600 or 800 units.

Pneumoperitoneum.—Roentgenograms were taken to demonstrate free air within the peritoneal cavity in 90.3 per cent of all cases, only those with erroneous diagnoses escaping. Within six hours after perforation 63.5 per cent had demonstrable free air, and after six more hours the x-ray views were posi-

TABLE XI. VARIATIONS OF PNEUMOPERITONEUM AND DURATION OF PERFORATION

DURATION OF PERFORATION (IN HOURS)	NUMBER OF PATIENTS	NUMBER WITH FREE AIR PRESENT	PER CENT
Less than 6	184	117	63.5
6 to 12	78	70	89.7
Greater than 12	35	30	85.7
Undetermined	26	16	
Total number of x-ray views	323	233	72.1

tive in 89.7 per cent. A slight decrease after twelve hours is possibly explained by resorption in a few cases.

An increase in mortality with the presence of free air was present, and was partially, if not wholly, explained by the increased mortality with the longer duration of perforation. The operative mortality increased from 14.8 per cent of those without free air, to 20.2 per cent of those in whom it was present.

Palpation.—Palpation of the abdomen within the first six hours revealed the familiar "board-like," involuntary rigidity in 98.6 per cent of cases, but it was not such a constant finding as time elapsed and the initial chemical peritonitis abated. After twenty-four hours it was present in less than one-half the cases (44.8 per cent); it was then often described as "voluntary" rigidity. By this time either distention was developing from ileus and bacterial peritonitis, or the peritoneal irritation was disappearing, apparently after a spontaneous sealing off of the perforation. Tenderness to some degree was almost always elicited.

Percussion.—Mention of liver dullness was found in comparatively few charts. Its obliteration usually corresponded to x-ray evidence of the larger amounts of free air in the peritoneal cavity.

Auscultation.—Auscultation for bowel sounds was carried out in two-thirds of the patients, and they were noted to be absent in 79.2 per cent of these. These findings were slightly more common within the first six hours (81.8 per cent). They were considered as being hypoactive in 13.8 per cent.

Errors in Diagnosis.—Including the diagnosis of "perforated viscus," a correct diagnosis of acute perforation was made in 321 of the 362 cases for an 11.3 per cent error for the disease. Of the 318 operative cases, the preoperative diagnosis was correct in 306 for a 3.8 per cent error for the disease. Acute appendicitis was the most common erroneous diagnosis, which was followed by acute cholecystitis.

Thirty-six patients without the disease were operated upon with a preoperative diagnosis of perforated ulcer. Therefore, the preoperative diagnosis of perforated ulcer was made correctly 306 times and incorrectly 36 times for a total of 342, a 10.5 per cent error in diagnosis. The most common disease mistaken for a perforated ulcer was acute appendicitis, and the next was acute cholecystitis.

TABLE XII. ERRORS IN DIAGNOSIS OF ACUTE PERFORATIONS

DIAGNOSES	NUMBER OF PATIENTS	PER CENT
Correct	321	88.7
Unsuspected	16	4.4
Acute appendicitis	11	3.0
Acute cholecystitis	5	1.4
In doubt	4	1.1
Intestinal obstruction	2	0.6
Carcinoma of stomach	2	0.6
Bleeding peptic ulcer	1	0.3
Total	362	100

TABLE XIII. OTHER DISEASES DIAGNOSED AS ACUTE PERFORATIONS

DISEASES	NUMBER OF PATIENTS	PER CENT
Correct	306	89.5
Acute appendicitis	7	2.0
Acute cholecystitis	6	1.8
No perforation	6	1.8
Acute pancreatitis	4	1.2
Traumatic rupture of bowel	4	1.2
Carcinoma of sigmoid with perforation	2	0.6
Foreign body perforation of ileum	2	0.6
Right lower lobe pneumonia	1	0.3
Mesenteric thrombosis	1	0.3
Intestinal strangulation	1	0.3
Peritoneal carcinomatosis	1	0.3
Twisted ovarian cyst	1	0.3
Total	342	100

Hours Before Operation.—Because of the severity of the pain, most of the patients entered the hospital early in the disease, and the diagnosis was made without difficulty in the majority. The younger patients were operated upon more promptly: 50 per cent of the teen age group, 40 per cent of the 20 year, 30 per cent of the 30 year, and only 20 per cent of the following age groups were operated upon within six hours after perforation. The increased mortality of the aged patient was partially explained by the delay in presenting himself and the usually less typical history and physical findings, all of which contributed to a longer time interval before diagnosis and operation. The mortality increased from 5.8 per cent when the perforation was of less than six hours' duration to 54.5 per cent, when the operation was performed after twenty-four hours.

TABLE XIV. NUMBER OF HOURS OF PERFORATION BEFORE OPERATION AND MORTALITY

NUMBER OF HOURS BEFORE OPERATION	OPERATIVE CASES	PER CENT	DEATHS	PER CENT OPERATIVE MORTALITY
Less than 6	86	27.0	5	5.8
6 to 12	160	50.3	22	13.7
12 to 24	47	14.8	19	40.4
Greater than 24	22	6.9	12	54.5
Indeterminate	3	0.9	0	
Total	318	100	58	18.2

Once the diagnosis was made and the patient was prepared with intravenous fluids or blood to tolerate surgery, an exploratory laparotomy was carried out. All operations were done by the house-officers, a total of 61 men during the ten-year period.

TABLE XV. VARIATIONS OF ANESTHESIA AND MORTALITY

ANESTHESIA	OPERATIVE CASES	DEATHS	PER CENT OPERATIVE MORTALITY
General	66	8	12.1
Spinal	198	28	14.1
Spinal plus general	40	11	27.5
Local	9	7	77.8
Local plus general	2	2	100.0
Unknown	3	2	
Total	318	58	18.2

Anesthesia.—The selection of anesthesia depended upon the patient's condition and the surgeon's choice of available anesthesia. A general or a spinal anesthesia, which did not need supplementation, produced about equal results. If, however, the spinal was inadequate in duration of time or level of anesthesia and a general anesthesia was found necessary for supplementation, the mortality rate was twice as high. Each has its advantage and disadvantage which cannot be discussed here, although it might be mentioned that endotracheal anesthesia is gaining in popularity at St. Louis City Hospital. The reason local anesthesia made such a poor record was undoubtedly because it had been reserved for the poorest risk patients.

The choice of incision varied with the particular surgeon's favorite incision, and with the degree of certainty of the diagnosis. When McBurney incisions were made because of an erroneous diagnosis, they were closed and epigastric incisions were made.

Upon entering the peritoneal cavity, varying amounts of free air and free fluid, the descriptions of which depended upon the food contents and duration of the perforation, were encountered.

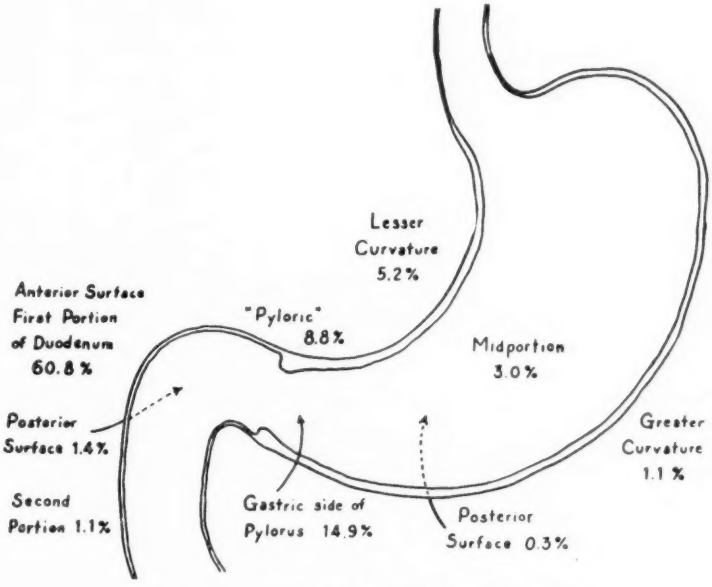

Fig. 5.—Locations of perforated peptic ulcers.

Location.—The majority, 60.8 per cent, of the perforations were on the anterior surface of the first portion of the duodenum. Because of the distortion of the anatomy from scarring and edema, many of those described as "pyloric" or as being just beyond the pylorus on the gastric side were actually in the duodenum. In several cases this error was corrected at autopsy or by biopsy which showed Brunner's glands. Perforations of gastric ulcers constituted 24.6 per cent. The operative mortality of the latter was

24.1 per cent as compared to 16.4 per cent for duodenal perforations. This may be partially explained by the fact that the gastric perforations tended to occur in older individuals. A few perforations of ulcers of the posterior surface into the lesser omental sac, of the second portion of the duodenum, and of a gastrojejunal stoma were encountered. There was one case in which two simultaneous perforations of gastric ulcers occurred. One perforation of a peptic ulcer in a Meckle's diverticulum was proved by excision of the diverticulum and demonstration of gastric glands. Two other cases of perforation in the terminal ileum were believed also to be from aberrant gastric mucosa. Of the cases, 2.2 per cent were perforations of carcinomas of the stomach, and their operative mortality was 80 per cent.

TABLE XVI. VARIATIONS OF LOCATION OF ACUTE PERFORATIONS AND MORTALITY

LOCATION	OPERATIVE CASES	DEATHS	PER CENT OPERATIVE MORTALITY	TOTAL CASES	PER CENT
Stomach					
Greater curvature	3	2	66.7	4	1.1
Lesser curvature	17	5	29.4	19	5.2
Mid-portion	8	5	62.5	11	3.0
Posterior surface	1	1	100.0	1	0.3
Gastric side of pylorus	54	7	13.0	54	14.9
Total gastric	83	20	24.1	89	24.6
"Pyloric"	32	1	3.1	32	8.8
Duodenum					
First portion, anterior	191	31	16.2	220	60.8
Posterior surface	1	1	100.0	5	1.4
Second portion	3	0	0.0	4	1.1
Total duodenal	195	32	16.4	229	63.3
Gastrojejunal stoma	1	0	0.0	1	0.3
Terminal ileum (Meckle's)	2	1	50.0	3	0.8
Carcinoma of stomach	5	4	80.0	8	2.2
Total	318	58	18.2	362	100

Size.—Perforations of gastric ulcers tended to be slightly larger than those in the duodenum, although the majority of both were less than 5 mm. in diameter. The operative mortality, however, was 14.4 per cent for the smaller perforations and 33.3 per cent for those larger than 1 cm.

Biopsies were taken in 20.8 per cent of all perforations and were positive for carcinoma in 75 per cent of the gastric carcinomas.

Treatment of Perforation.—Most of the perforations were treated with simple closures, and these were effected in three ways: purse-string suture, interrupted sutures, both either with or without reinforcement with omentum, and simple tying of omentum over the perforation as a free or living omental graft. The operative mortality following the latter closure was only 6.9 per cent. The few remaining methods of treatment included pyloroplasty, gastrojejunostomy, and subtotal gastrectomy, but these were in selected cases and were too few in number to permit any conclusions.

TABLE XVII. METHODS OF TREATMENT OF PERFORATION AND MORTALITY

TREATMENT	OPERATIVE CASES	PER CENT	DEATHS	PER CENT OPERATIVE MORTALITY
Simple				
Purse-string suture	136	43.2	24	17.6
Interrupted sutures	78	24.8	19	24.4
Omental graft	87	27.6	6	6.9
Total simple closures	301	95.6	49	16.3
Simple closure of carcinoma	5	1.6	4	80.0
Pyloroplasty	2	0.6	1	50.0
Subtotal gastrectomy	2	0.6	0	0.0
Simple closure plus gastrojejunostomy	2	0.6	1	50.0
Purse-string suture of ileum	1	0.3	1	100.0
Excision and suture of Meckle's	1	0.3	0	0.0
Perforation not found at laparotomy	1	0.3	1	100.0
Total known	315	100	57	18.2

Culture.—Cultures of the peritoneal fluid were taken at operation in two-thirds of the cases, and 42.5 per cent were reported as sterile or as growing yeast or fungus, which were considered contaminants. The operative mortality in these cases was 7.7 per cent, while it was 24.3 per cent for the 30.2 per cent in which streptococci were cultured, and 54.5 per cent for the 4.5 per cent in which *Bacillus coli* were cultured. Cultures from perforations older than twelve hours were more likely to be positive than those less than twelve hours. *B. coli*, the only organism not found normally in the respiratory and upper gastrointestinal tract, was found five times more often in the older perforations.

TABLE XVIII. VARIATIONS OF CULTURE, DURATION OF PERFORATION, AND MORTALITY

CULTURE	NUMBER LESS THAN 12 HOURS	PER CENT	NUMBER MORE THAN 12 HOURS	PER CENT	TOTAL	PER CENT	DEATHS	PER CENT
Sterile	89	49.4	15	23.1	104	42.5	8	7.7
Diphtheroids	10	5.5	2	3.1	12	4.9	2	16.6
Staphylococci	25	13.9	12	18.5	37	15.1	7	18.9
Streptococci	50	27.8	24	37.0	74	30.2	18	24.3
Pneumococci	2	1.1	5	7.7	7	2.9	2	28.6
B. coli	4	2.2	7	10.8	11	4.5	6	54.6
Total	180	100	65	100	245	100	43	17.5

TABLE XIX. VARIATIONS OF WOUND HEALING, DRAINAGE, AND DURATION OF PERFORATION

DRAINAGE	NUMBER OF PATIENTS	PER CENT	NUMBER INFECTED	PER CENT	NUMBER DISRUPTED	PER CENT
Closed without drainage	168	59.8	32	19.1	4	2.3
Drainage through stab wound	94	33.4	34	36.2	3	3.2
Drainage through incision	19	6.8	9	47.4	2	10.5
DURATION OF PERFORATION (IN HOURS)						
Less than 6	83		13	15.7	1	1.2
6 to 12	148		39	26.3	5	3.4
12 to 24	35		15	42.9	2	5.7
Greater than 24	15		8	53.3	1	6.7
Results of healing known	281	100	75	26.7	9	3.2

Wounds.—All incisions are now being closed without drainage, except when localized pus is present. Several years ago, however, the majority were drained, mostly through a stab wound in the right flank. Of all the operative cases, 40.2 per cent were drained, and in these the percentage of wound infections was doubled. Wound disruptions were most common when the drain was brought out through the wound. Wound infections increased with the duration of perforation, increasing from 15.7 per cent within the first six hours to 53.3 per cent after twenty-four hours.

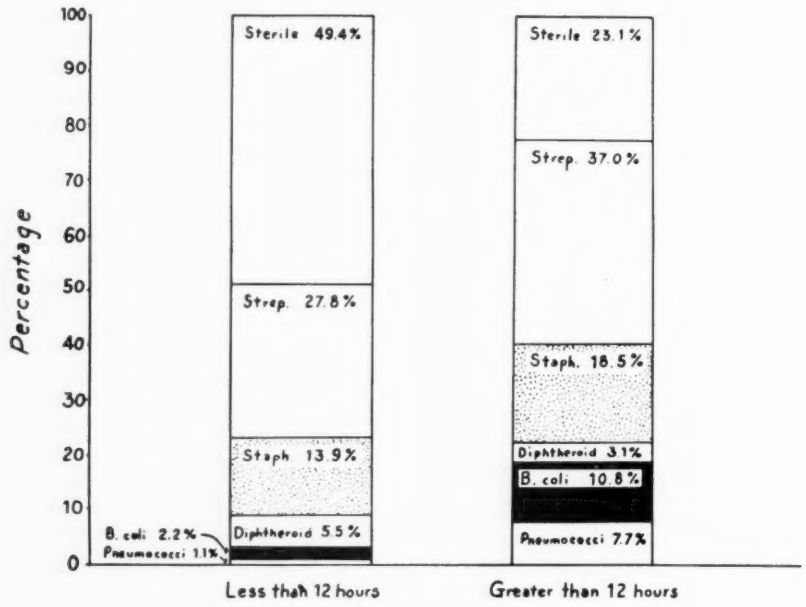

Fig. 6.—Cultures and duration of perforation.

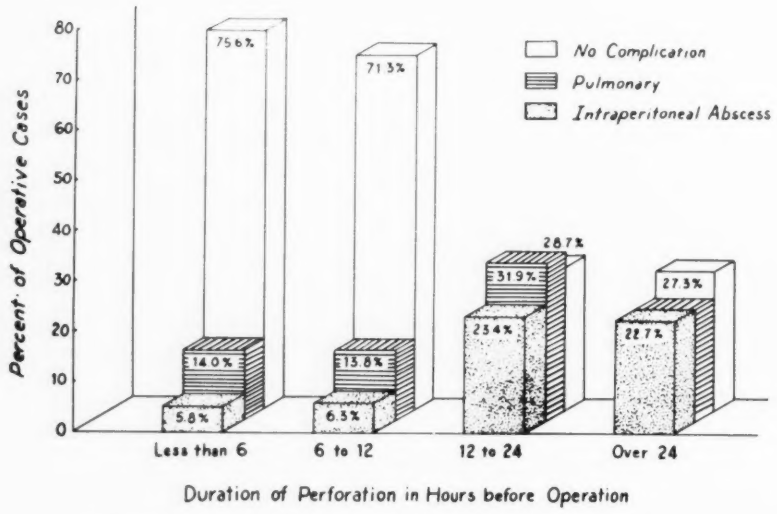

Fig. 7.—Common complications and duration of perforation.

Complications.—Postoperatively the patients were maintained on parenteral fluids and continuous Wangensteen suction. Antibiotics and other medications were variably administered according to necessity. Excluding wound infections, 62.3 per cent of the operative cases were uncomplicated. When the patients were operated upon within six hours, 75.6 per cent were uncomplicated, and after twenty-four hours, only 27.3 per cent. Persistent purulent peritonitis was considered a complication when it was believed to have caused the death of the patient, otherwise peritonitis was not considered a complication, since it was present in all to varying degrees. Pulmonary complications were most common, having occurred in 17 per cent. Undoubtedly, there were many more in the form of small pleural effusions and atelectases which were undiagnosed. Intraperitoneal abscesses developed in 5.8 per cent of the patients operated upon within six hours, and in 22.7 per cent of those operated upon after twenty-four hours. In these cases 88.5 per cent of the peritoneal fluid cultures taken at the time of operation were positive. There were, however, no complications in 51.9 per cent of the positive cultures, but 22.1 per cent of them did develop intraperitoneal abscesses. Of the patients who developed subdiaphragmatic abscesses, 65 per cent had had drainage at the time of operation.

TABLE XX. VARIATIONS OF COMPLICATIONS AND MORTALITY

COMPLICATIONS	OPERATIVE CASES	PER CENT	DEATHS	PER CENT MORTALITY
None	198	62.3		
Pulmonary	54	17.0	19	35.2
Intraperitoneal Abscesses				
Subphrenic	17	5.3	8	47.1
Right gutter	3	0.9	2	66.7
Pelvic	11	3.5	2	18.2
Total intraperitoneal abscesses	31	9.7	12	38.7
Persistent purulent peritonitis	29	9.1	29	100.0
Wound disruption	9	2.8	2	22.2
Delirium tremens	4	1.3	0	0.0
Uremia	4	1.3	3	75.0
Parotitis	3	0.9	2	66.7
Pyloric obstruction requiring gastroenterostomy	3	0.9	1	33.3
Exsanguination	3	0.9	3	100.0
Cardiac decompensation	2	0.6	1	50.0
Cirrhosis of liver	1	0.3	1	100.0
Undetermined	7		4	
Total number of complications	348		77	
More than one complication	30	9.4	19	63.3
Total	318	100	58	18.2

Postoperative Studies.—During their convalescence, 84 patients were examined with a barium meal, and the ulcer was demonstrated in 86.9 per cent of them. Sixty-three patients were subjected to gastric analyses and only 39.7 per cent were found to have hyperacidity at that time.

Follow-up: Unfortunately, the follow-up of most of the 260 surviving patients is poor and entirely missing in many. Subtotal gastrectomies have been performed subsequently for various reasons in 30 of the cases, with a post-

operative mortality of 13.3 per cent. An attempt will be made to contact as many of these patients as possible, and to report at a later date a summary of their present gastrointestinal conditions.

SUMMARY

In this report 362 consecutive cases of acute perforation of stomach and small bowel ulcerations, occurring during the past ten years at St. Louis City Hospital, have been summarized. Of these, 318 patients were treated surgically, and the operative mortality was 18.2 per cent.

More perforations occurred during the spring and fewer during the winter, when the mortality was twice as high. Between 3 and 6 P.M. was the most common time of day for the operation to occur, and 5 A.M. was the most uncommon.

The majority of the patients (95.3 per cent) were men. Patients between 40 and 50 years of age were most prevalent. The mortality increased with age from 0 per cent in the teens to 73.7 per cent in the 70-year age group.

Histories of symptoms referable to the upper gastrointestinal tract were obtained in 85.4 per cent, and there was no history in the remainder. When present, the duration of symptoms was equally divided among less than one year, between one and five years, and longer than five years. In 24.6 per cent there was a history of either hematemesis or melena.

The pain of the majority was severe and sudden in onset, and was epigastric in 90.9 per cent. Referral of pain to the shoulder was present in 65.8 per cent of patients. The perforation was accompanied by nausea and vomiting in 63.2 per cent; 5.8 per cent vomited in varying amounts.

The admission temperatures, pulse, and respiratory rates were relatively normal in early perforations, but were found to increase with the duration of time after perforation.

Most white blood cell counts were between 10,000 and 20,000. The mortality increased from 12.3 per cent of those with a leucocytosis over 20,000 to 75 per cent of those with a leucopenia less than 5,000.

Diastase activity was relatively normal in all instances.

Pneumoperitoneum was demonstrable in 72.1 per cent of cases.

Involuntary abdominal rigidity was present in 98.6 per cent of the early perforations but was present in less than one-half of those over twenty-four hours old. Bowel sounds were absent in 79.2 per cent of the cases and hypoactive in most of the remainder.

The most common erroneous diagnosis was acute appendicitis, followed by acute cholecystitis.

Of the patients, 77 per cent were operated upon within twelve hours after perforation. The mortality increased from 5.8 per cent, when within six hours, to 54.5 per cent, when after twenty-four hours.

Best results were obtained with general anesthetics, but an adequate spinal was found equally satisfactory.

Of the perforations, 60.8 per cent occurred in ulcers on the anterior surface of the first portion of the duodenum, and 24.6 per cent occurred in the stomach.

Simple closure of the perforation with an omental graft was the most satisfactory method of treatment, the mortality of these patients having been only 6.9 per cent.

Within twelve hours after perforation, one-half of cultures of the peritoneal fluid were sterile, but as time elapsed the positive cultures increased in number.

Of the wounds closed with drainage, 36.2 per cent became infected, while 19.1 per cent of those closed without drainage became infected. Wound infections rose from 15.7 per cent after early operations to 53.3 per cent following operations after twenty-four hours.

Excluding wound infections, pulmonary complications were most common, having occurred in 17 per cent of the operative cases. Intraperitoneal abscesses developed in 9.7 per cent of the patients and were more common when an attempt had been made to drain the peritoneal cavity, and when the operation had been performed after twelve hours.

CONCLUSIONS

The duration of time from perforation to operation seemed to be the most important factor in influencing the prognosis. As time elapsed, increased soilage of the peritoneal cavity and bacterial infection occurred, and dehydration and electrolyte imbalance increased. Therefore, the most effective treatment consisted of correction or prevention of electrolyte imbalance and surgery as soon as possible to close the perforation with an omental graft and to close the incision without drainage.

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RESTORATION OF GRASPING FUNCTION FOLLOWING LOSS OF ALL FIVE DIGITS

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LOSS of all five digits in one or both hands due to freezing, burn, or other trauma is an extremely crippling deformity. Some grasping function of the hand may be restored by the elongation of the thumb and the formation of a cleft between the stumps of the thumb and the middle finger, that is between the first and third metacarpals. This is dependent on there being movement at the articulation of the first metacarpal with the greater multangular bone.

TECHNIQUE

Closure of the unhealed stump of the hand is done with either a free skin graft or a direct flap. If already healed with scar epithelium a flap may still be necessary for better coverage, but the skin of the palm and dorsum may be sufficiently loose to allow closure over the end. As in all deep work success depends on having surface tissue that is capable of firm and permanent healing.

Elongation of the thumb is indicated if the bony stump of the thumb is shorter than that of the index finger. To do this, the head of the second metacarpal is transplanted over the end of the first metacarpal. This sometimes increases the length of the thumb by 2 cm. The procedure is carried out through a dorsal incision so placed that it can be used again at the time the cleft is created. The shaft of the second metacarpal is cut across a little proximal to the level of the end of the first. The reminder of the shaft is removed leaving the base to prevent collapse of the metacarpal arch. After carefully dissecting off the skin from the stump of the first metacarpal, the end of the bone is squared off, freshened, and displaced over in line with and behind the head of the second metacarpal. The bones are fixed in position with minimal angulation using a Kirschner wire passed through the end of the second metacarpal into the shaft of the first.

The formation of a cleft is the final procedure and is done after there is good bony union. The skin between the first and third metacarpals is opened preserving a dorsal rectangular flap with the base proximally. By blunt dissection of the tissues in the cleft, the nerves can be saved and the thenar muscles displaced backward. The flap is then sutured across the base of the cleft and the raw surfaces on the sides covered with a thick split free skin graft.

If the bony elongation is not done, as for example when there is no extra length to gain from the second or when there remains a short stump of thumb, the cleft is made in a single stage removing the second metacarpal at the same time.

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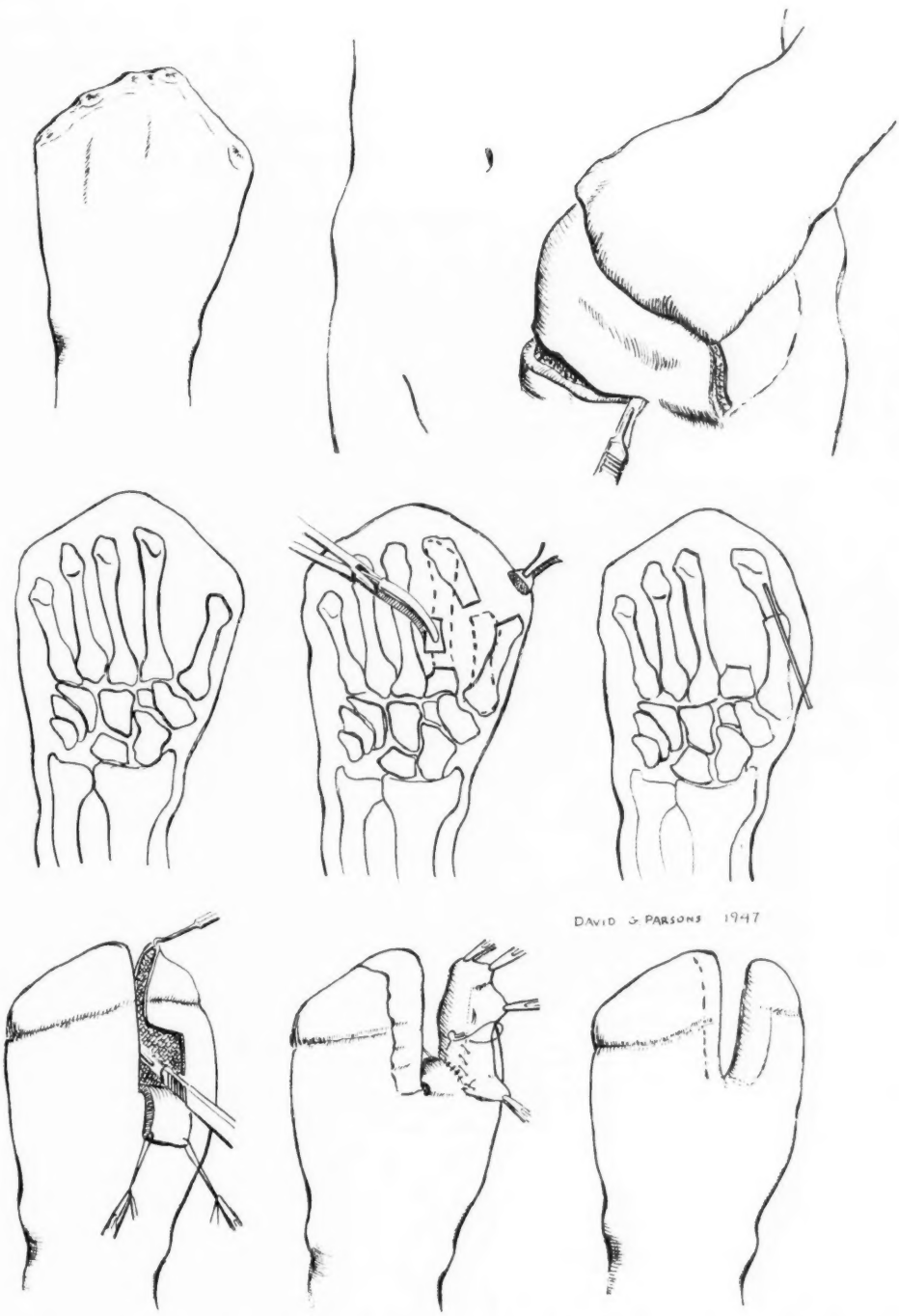

Fig. 1.—Stump of hand resurfaced with direct abdominal flap. Head of second metacarpal was transplanted over the end of the first and held with a Kirschner wire. Clefting of the hand was completed with direct flap transposed across the base of the cleft and free skin grafts to cover the sides.

COMMENT

Long ago (1837) Sir Charles Bell said that the child at the zoo marveled at the working of the elephant's trunk, but that he could better look down at his own hand and marvel about the approximation of the thumb and the fingers. Their function was said to be the most distinguishing human anatomical feature. In restorative surgery of the hand, the pioneer and continued work of Kanavel,

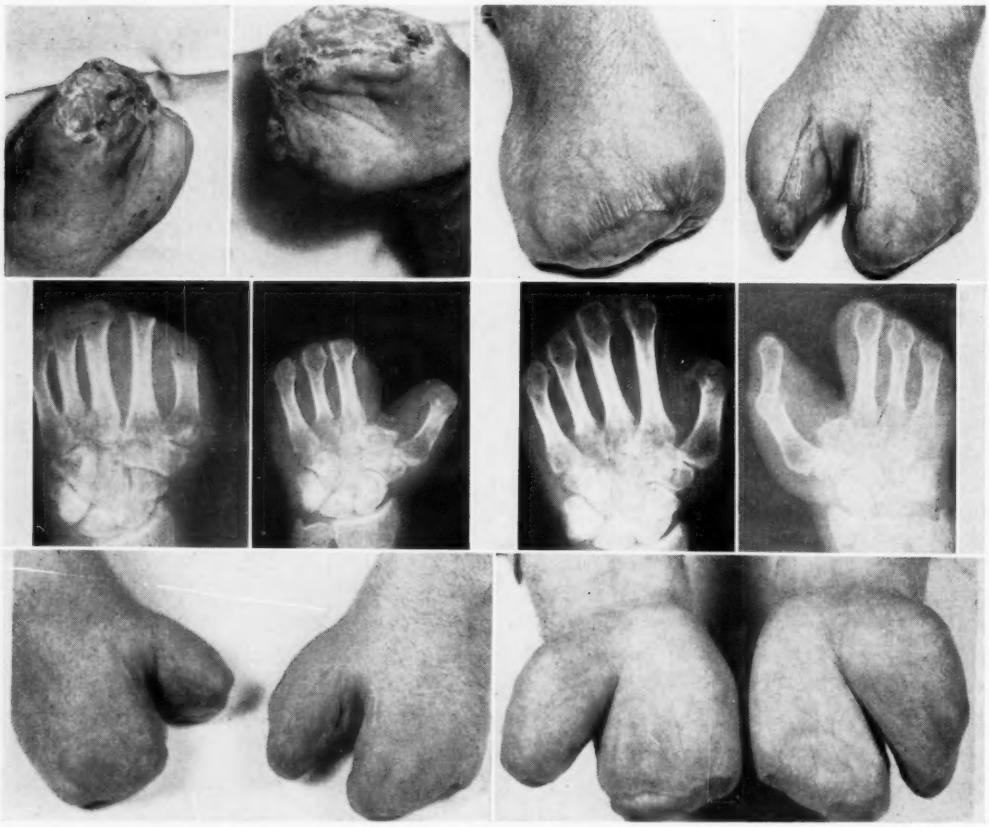

Fig. 2.—High altitude freeze. All fingers and both thumbs were lost. Since the patient was practically blind from freezing of face and eyes, restoration of grasping function and preservation of sensation were most important. Unhealed stumps of hands were dressed in free skin grafts. Grafts were later excised and the skin of the palm and dorsum was closed over the ends. The left thumb was elongated by transplanting the second metacarpal head preliminary to cleft-ing of the hand. Clefting of the right hand was completed by excising the shaft and head of the second metacarpal without preliminary elongation of the thumb since the first and second metacarpal were of almost equal length.

Koch, Mason, and Bunnell will always provide a stimulus to workers in this important phase of surgery. The restorative procedure described here is an example of substitution by surgery for a lost function; the results are crude in comparison with the original but supply an element of that "most distinguishing anatomical feature."

By this method good motion of the thumb and development of considerable strength can result. The hands are converted from ineffectual stumps into useful members for grasping and for holding utensils, tools, etc. No longer must the person be fed, dressed, and depend completely on other hands. Also

by this method normal sensation in the functioning part of the hand is preserved whereas elongation of a digit by a tubed flap results in an anesthetic stump which may or may not regain sensation. By utilizing the head of the second metacarpal to elongate the first and by later creating a cleft between the first and the third metacarpals, an anesthetic tubed flap is unnecessary. Sensation develops in a matter of weeks in the skin grafts, but takes longer when a flap is used to cover the end of the hand. There has been no change in sensation observed in the normal skin following operation.

Fig. 3.—High altitude freeze. All fingers and thumb of the left hand and several fingers of the right hand were lost. Scar epithelium on the stump of the left hand was replaced with direct abdominal flap. Open stumps of fingers of the right hand were closed with skin grafts. Adhesions between fingers were severed and grafted. The left thumb was elongated by transplanting the second metacarpal head preliminary to clefting of the hand.

SUMMARY

A method for restoring an effective grasping function to a hand crippled by the loss of all five digits is presented.

Elongation of the thumb and clefting of the hand are accomplished without disturbing normal sensation in the functioning part of the hand.

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THE DIAGNOSIS OF LESIONS OF THE BREAST

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THE recent widespread dissemination of information about cancer, by press, radio, and pamphlet is making the layman increasingly "cancer conscious." A growing number of patients are presenting themselves to physicians with early cancer or with benign lesions suggestive of cancer for which they need treatment or reassurance. One of the organs most frequently concerned is the breast, where pain is common and where lumps can be readily discovered by the patient.

It is probable, therefore, that the average physician's practice today contains a larger percentage of patients with early cancer of the breast and small benign lesions suspicious of cancer than it did five years ago. Such a shift in the incidence of various breast lesions increases the physician's difficulties. He must advise a growing number of apprehensive patients with questionable lesions not easily diagnosed by history and physical examination. He must confidently reassure a growing number of patients with nonexistent lesions who have developed cancer phobia. How accurate are the present diagnostic techniques for breast lesions?

We have reviewed the records of 250 consecutive patients with breast lesions, not due to infection, referred for surgical consultation to the senior surgeon of the Hospital of the University of Pennsylvania during 1946 and 1947. They should constitute a representative sample of patients with breast lesions referred by their physicians for a surgical consultation. All operations were performed by the senior surgeon or by one of us, and all pathologic examinations were made by Doctor Robert C. Horn, the surgical pathologist of the hospital.

Our objective in this study was to answer the following questions:

1. What is the present incidence of various types of breast lesions in a representative group of patients referred to a surgeon?
2. What is the incidence of early as compared to late cancer in such a group?
3. How accurately can lesions of the breast be diagnosed by (a) physical examination, (b) gross examination at the operating table, and (c) frozen section?
4. What are the early signs of carcinoma of the breast?
5. Is aspiration of breast cysts a safe diagnostic and therapeutic procedure?

Incidence of Breast Lesions Encountered.—The incidence of the various types of breast lesions found in our group of 250 patients is shown in Table I. Approximately one-fifth had carcinoma, a higher percentage than would be seen by the average practitioner since our group had been screened by referring

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TABLE I. FINAL DIAGNOSIS MADE IN 250 PATIENTS WITH BREAST LESIONS

DISEASE	NUMBER	PERCENTAGE
Chronic cystic mastitis	145	58.0
Carcinoma	48	19.2
Fibro-adenoma	21	8.4
Gynecomastia	12	4.8
Intraductal papilloma	9	3.6
Miscellaneous	15	6.0
Total	250	100.0

physicians. More than one-half the patients had chronic cystic mastitis. Many of these were observed on physical examination to have only slight abnormalities, but the diagnosis was strengthened by a typical history of pain in the breasts during the menses. No attempt was made to divide chronic cystic mastitis into the several categories used by some observers. One-fifth of the patients with chronic cystic mastitis had cancer phobia, and much of their physical and mental pain disappeared when they were convinced they did not have cancer.

The average age of the patient was determined for the three largest groups: chronic cystic mastitis, cancer, and fibro-adenoma. Patients with fibro-adenoma averaged 30 years; with chronic cystic mastitis, 40 years; and with cancer, 50 years.

Only twelve patients were males. Nine were boys in puberty or adolescence who had generalized hypertrophy of one or both breasts without other obvious disease. They were reassured and told that no operation was required unless for cosmetic reasons. Three were men over 60 years with hypertrophy. Operation was performed on two in order to rule out cancer.

Physical Findings.—In formulating our ideas about the diagnosis of breast lesions, we have found the concept of the "dominant lump" helpful. This term is used by Stout* to designate a circumscribed area in the breast different in consistency from the rest of the mammary tissue. It takes considerable practice to differentiate small thickened or shotty areas from a true dominant lump, but with experience the examiner is able to locate them with confidence.

Among the 250 patients of this series, 170 dominant lumps were discovered. Patients without dominant lumps included many with diffuse chronic cystic mastitis, several with hypertrophy of the breast, and a few with intraductal

TABLE II. PHYSICAL CHARACTERISTICS OF THREE TYPES OF DOMINANT LUMPS*

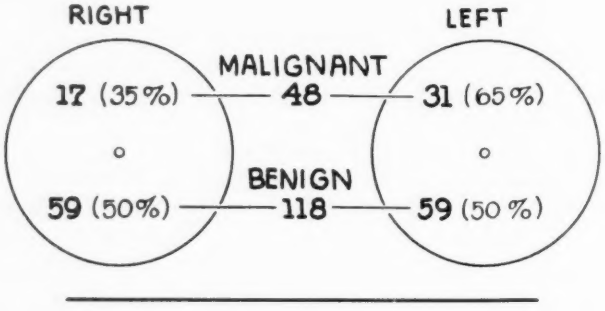
DISEASE	NO.	HARD	FIRM	IRREGULAR	"SLIPPABLE"	FIXED TO SKIN	EDEMA OF SKIN	TENDER
Chronic cystic mastitis (exclusive of cysts treated only by aspiration)	65	12	50	60	2	4	0	8
Cancer	48	39	5	28	2	24	9	0
Fibro-adenoma	21	11	10	1	13	1	0	1
Total	134	62	65	89	17	29	9	9

*Final diagnosis by pathologic examination. The characteristics noted are the most striking ones. Totals for each category do not equal the total number of cases.

*Stout, A. P.: The Technique of Diagnosis of Breast Carcinoma, the Choice of Treatment, and the Criteria of Inoperability, Homer G. Phillips Hospital, Tumor Staff meeting, J. Nat. M. A. 39:94-100, 1947.

papillomas and miscellaneous lesions. Patients with a dominant lump were routinely subjected to operation, and the final diagnosis was made by pathologic examination, except in those instances where the aspiration of clear fluid and subsequent collapse of the mass afforded definite evidence of a simple cyst.

Table II shows the physical characteristics associated with dominant lumps of three principal types: chronic cystic mastitis, cancer, and fibro-adenoma. These dominant lumps total 134 and do not include the breast cysts and miscellaneous dominant lumps. Most of the cancers were described as hard on physical examination, while the benign lesions were usually called firm. Almost one-half of the cancers were seen before they had become fixed to the skin or deep tissues and before edema could be demonstrated. Only one cancer had produced ulceration of the skin. A helpful diagnostic sign of the fibro-adenoma was its "slippability," found in over one-half. Carcinomas and fibro-adenomas were characteristically nontender.



By quadrants:

	MALIGNANT	BENIGN
1 Upper outer	28 (58%)	66 (56%)
2 Upper inner	9 (19%)	32 (27%)
3 Lower outer	4 (8%)	11 (9%)
4 Lower inner	7 (15%)	9 (8%)
Totals:	48	118

Fig. 1.—Location of 166 dominant lumps of the breast.

The location of the dominant lumps is given in Fig. 1. Sixty-five per cent of the malignant lesions were in the left breast, whereas the benign lesions were equally divided. The upper outer quadrants contained over one-half of both benign and malignant lesions.

A bleeding nipple was encountered in 14 of the 250 patients. Although 9 of these were found to have intraductal papillomas of at least microscopic proportions, in only 2 was the lesion discovered by gross pathologic examination. Of the patients with a bleeding nipple, 5 (36 per cent) had carcinoma.

Simple Cysts of the Breast.—The proper treatment of simple cysts of the breast is controversial. Early in the study we usually advised operation when the diagnosis of cyst was made. We later introduced the policy of attempting

aspiration of lumps which were thought to be cysts. We found that breast cysts large enough for aspiration were rubbery in consistency and smooth in outline. Several were tender. When a previous operation had shown cystic disease, we felt reassured in resorting to aspiration instead of exploratory operation. The clinical diagnosis of breast cyst seemed definite enough in 30 patients to warrant an attempt at aspiration. From 8 of these no fluid could be obtained, and the patients were operated upon. In 1 patient a carcinoma was found at operation. The remaining 7 patients with "dry aspiration" had benign lesions: 4, chronic cystic disease without gross cyst formation, and 3, deep-seated fibroadenomas. In 22 instances the dominant lump was collapsed by the aspiration of clear fluid (average amount 11.0 c.c.). Bloody fluid, which we would have considered an absolute indication for operation, was obtained in none.

Twenty of the 22 patients in whom aspiration was done have been followed at frequent intervals until the present time, for an average of eleven months. In 2 patients the cyst filled again, and a second aspiration was required. In the remaining 18 patients no recurrence has been noted. Two of our patients were lost to follow-up although strongly urged to return for repeated breast examinations.

TABLE III. ACCURACY OF PREOPERATIVE DIAGNOSIS OF 144 "DOMINANT BREAST LUMPS" (CORRECT IN 108 INSTANCES OR 75 PER CENT)

FINAL DIAGNOSIS	TOTALS	PREOPERATIVE DIAGNOSIS				PER CENT CORRECT
		CHRONIC CYSTIC MASTITIS	CARCINOMA	FIBRO-ADENOMA	MISC.	
Chronic cystic mastitis	65	47	8	9	1	72
Carcinoma	48	5	38	2	3	79
Fibro-adenoma	21	3	1	17	0	81
Miscellaneous	10*	2	2	0	6	60
Total	144	57	49	28	10	75

*Fat Necrosis, 2; galactocele, 1; lipoma, 4; sebaceous cyst, 3.

Accuracy of Preoperative Diagnosis.—We found that it was usually difficult and often impossible to diagnose a small cancer of the breast on physical examination. Many errors were made preoperatively in this series of patients. Table III offers a comparison of the preoperative and final diagnoses made in 144 dominant lumps. Approximately one-fourth were diagnosed incorrectly on physical examination. Of 48 malignant lesions, 10 were diagnosed preoperatively as benign. A common error was in diagnosing a small cancer as a fibroadenoma. On three occasions a dominant lump diagnosed lipoma was found at operation to be a small carcinoma embedded in fatty tissue. If one adheres to the rule of operating on all dominant lumps, a mistake in the preoperative diagnosis is not a serious one.

Accuracy of Diagnosis at Operation.—In contrast to the frequent inaccuracy of preoperative diagnosis, a diagnosis made at the operating table by gross examination and frozen section was almost always correct (Table IV).

In every case where gross examination left the surgeon or surgical pathologist in doubt as to the nature of a lesion, quick frozen section examination yielded sufficient information to make a correct diagnosis. Radical mastectomy was performed in two cases on the basis of a gross pathologic diagnosis although the frozen section verdict was equivocal. Both tumors were subsequently proved to be carcinomas.

It is often impossible to determine the presence of carcinoma in axillary lymph nodes by physical examination unless the nodes are extensively involved. Axillary lymph node metastases were frequently overlooked even on gross examination (Fig. 2). Of the 48 patients with carcinoma of the breast, 23 (47.9 per cent) were proved to have axillary metastases. Only one-half of these were recognized preoperatively; another one-quarter had metastases of sufficient extent to be recognized at the operating table. Just as many incorrect diagnoses were made when the axillary nodes were palpably enlarged by a nonmalignant process. Of 22 patients thought preoperatively to have axillary metastases, only one-half were shown to have lymph node involvement by subsequent study.

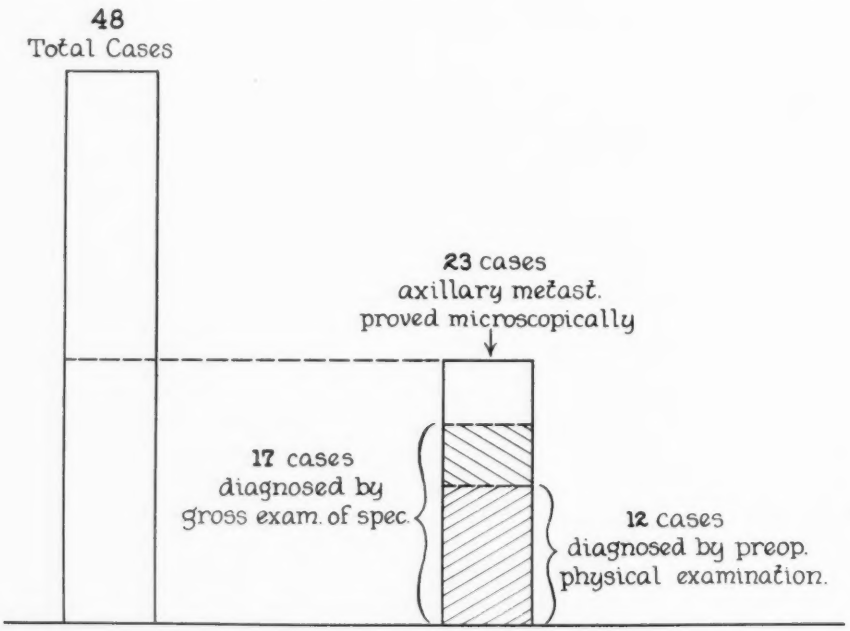


Fig. 2.—Diagnosis of axillary lymph node metastasis in 48 cases of carcinoma of the breast.

Comment.—An inevitable result of the recent campaigns to educate the layman about cancer is that women become breast conscious. An increasing number who have no serious lesion are consulting physicians because of a fear of breast cancer. The problem of convincing these women that they do not have cancer is great. The patient who is not completely reassured shops uselessly from physician to physician.

Of even greater importance is the early detection of cancer and its cure by radical mastectomy. The key to early diagnosis does not lie in a search for

TABLE IV. ACCURACY OF DIAGNOSIS AT OPERATION OF 144 "DOMINANT BREAST LUMPS"

FINAL PATHOLOGIC DIAGNOSIS	TOTALS	CHRONIC CYSTIC MASTITIS	CANCER	FIBRO-ADENOMA	MISC.	PER CENT CORRECT
By Gross Examination at the Operating Table (144 Examinations)						
Chronic cystic mastitis	65	64	0	1	0	98
Cancer	48	1	47	0	0	98
Fibro-adenoma	21	0	0	21	0	100
Miscellaneous	10	1	0	0	9	90
Total	144	66	47	22	9	
By Frozen Section (74 Examinations)						
					Undetermined	
Chronic cystic mastitis	28	27	0	0	1	96.4
Cancer	41	0	39	0	2	95
Fibro-adenoma	5	0	0	5	0	100
Total	74	27	39	5	3	

the "textbook" signs of malignancy, for example, skin retraction, edema, axillary metastases, etc., which may not appear until the disease has become incurable, but in the recognition of small and often indefinite dominant lumps. Whenever an area of thickening is localized, it must be re-examined at frequent intervals. If it develops the characteristics of a dominant lump, its nature must be determined by surgical removal and pathologic study, unless the aspiration of clear fluid with subsequent complete collapse shows it to be a simple cyst, when under strict criteria operation may be deferred. Our experience indicates that the correct diagnosis of a mass in the breast can be made preoperatively in only about three-fourths of the cases. By gross and frozen section examination at the operating table, however, a correct diagnosis can be made in practically 100 per cent.

We do not advise the routine use of aspiration for suspected cysts: first, because of the possibility that the patient may not return for further examination and, second, because of the possibility of spreading an unrecognized cancer by the procedure. The rare patient with both carcinoma and a cyst may be led to believe the lesion has been cured and may not return for re-examination until the carcinoma has metastasized. In one of our patients aspiration was attempted in the presence of carcinoma and could conceivably have spread the cancer although radical mastectomy was performed within a week. Nonetheless, the benefits of aspiration were usually great, especially in the dramatic relief afforded several women with cancer phobia. If aspiration is performed, a meticulous follow-up must be insisted upon, and the procedure must be performed only by those experienced in the clinical diagnosis of breast lesions. Attempts should be made to study the aspirated fluid by the Papanicolaou technique if trained personnel are available. In all instances in which the mass does not completely disappear by the aspiration of fluid, in all instances of "dry tap," and in all instances in which the fluid is bloody, the lesion must be excised for pathologic study.

CONCLUSIONS

1. A study of 250 patients with breast complaints referred for a surgical consultation by their examining physicians showed that one-fifth had carcinoma and slightly more than one-half had diagnoses of chronic cystic mastitis.

2. Forty-eight per cent of the patients with carcinoma were found to have axillary metastases.

3. In the patients operated upon, the correct diagnosis of a lesion of the breast was made preoperatively in only 75 per cent of patients, whereas it was made at operation by combined gross inspection and frozen section examination in 100 per cent.

4. In the clinical examination of the breast the ability to recognize small and elusive dominant lumps is of much greater importance than the ability to diagnose the mass correctly. Dominant lumps must be regarded as cancer and explored surgically.

5. Aspiration of breast cysts may be a useful procedure when practiced under rigid criteria. It is not recommended as a procedure for general adoption.

DOUBLE GALL BLADDERS

A Report of Four Cases, One With Suppurative Cholangitis and Bacteremia

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A TRUE "double" gall bladder denotes a complete duplication of the vesica fellea. There must be two separate and distinct gall bladders, each of which is drained by its own cystic duct. The manner in which the cystic ducts unite with the common hepatic and choledochal bile ducts can and does vary. Usually the two separate cystic ducts empty into the same segment of the extrahepatic ductal system, but occasionally one duct unites with the choledochus while the other fuses with the common hepatic duct. At times, however, the paired cystic ducts may fuse to form a common cystic duct before joining the common hepatic bile duct. The latter is designated as the Y type.

The double gall bladders must not be confused with the "bilobed" or "bifid" varieties. In the bilobed type there is no duplication of the gall bladder itself, but rather a division of its cavity into two compartments by a longitudinally placed membranous septum. Strangely enough, this septum separates the fundal and corporeal segments of the lumen into two distinct compartments, but at the neck of the gall bladder the dividing septum disappears, thus forming a common chamber which is drained by a single cystic duct. In the bifid type, the fundal segment of the gall bladder bifurcates, thus forming two diverticular pockets which unite in the main gall bladder cavity to form a single structure.

As a rule, the double gall bladders are not contiguous. The accessory structure may occupy several unusual positions. It has been found hidden between the leaves of the gastrohepatic ligament or attached to the ventral surface of the left lobe of the liver.[2] Crile[1] found an accessory gall bladder completely buried within the substance of the right hepatic lobe. When the two gall bladders are contiguous, they may have a common peritoneal investment, giving them an appearance of a single structure.

DOUBLE GALL BLADDER: STONES IN GALL BLADDER AND EXTRAHEPATIC BILE DUCTS; SUPPURATIVE CHOLANGITIS AND STREPTOCOCCAL BACTEREMIA

Case 1.—A housewife, 40 years of age, was admitted to the Latter Day Saints Hospital, on Feb. 16, 1947, because of "gall bladder trouble." During the eight months prior to admission, she had had recurrent episodes of pain in the right subcostal area, accompanied by nausea, vomiting, and diarrhea. Each attack persisted for four days and was always followed by a state of well being. The paroxysm began seven days prior to hospitalization and was characterized by typical pains of gallstone colic. No history of jaundice could be obtained.

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Physical examination disclosed definite tenderness in the right subcostal zone. The liver and gall bladder could not be palpated. The gall bladder was not visualized by cholecystographic studies. The icteric index was 10; the stools contained bile, and the urine was acholic.

When the abdomen was opened, the gall bladder was found to be adherent to the stomach, colon, and omentum. The gall bladder contained many small calculi and its walls were indurated and fibrotic. Since nothing was seen to denote any anatomic abnormalities, a simple cholecystectomy was performed.

Fig. 1.—Forty-five cubic centimeters of 35 per cent Diodrast was injected into the external biliary fistula and an x-ray film was taken. Note the dilation of the right and left hepatic ducts. The common hepatic duct presents a mottled appearance because the intraductal stones partially displace the contrast medium. An obstructive lesion prevented the Diodrast from flowing down along the choledochus.

The pathologist described the excised tissues as follows. The specimen consisted of a gall bladder measuring 9 by 3 cm. Its mucosal surface was somewhat scarred and the wall had undergone considerable fibrosis. The mucosa was studded with deposits of cholesterol which gave it a yellowish discoloration. The gall bladder cavity contained thirty small faceted stones, with an average diameter of 1.5 cm. Microscopic studies failed to show any evidence of malignancy.

The postoperative convalescence was uneventful except that bile continued to flow from the drainage tract. Since it was hoped that the external biliary fistula might close spontaneously, the patient was permitted to return home on the eighth postoperative day. The drainage from the fistula continued for three days and then ceased abruptly. Subsequently the patient began to complain of abdominal distress, headache, nausea, and severe colicky pains. During this period the stools were acholic, the sclera became icteroid, and a most distressing pruritus plagued the patient. Four days later the fistula reopened and large quantities of dark bile escaped. The itching and jaundice persisted even though the abdominal cramps had subsided.

Two weeks after the primary operation, when the patient was seen in consultation, visualizing cholangiograms were obtained by injecting 35 c.c. of Diodrast into the external fistulous opening. The resulting x-ray picture demonstrated that the draining sinus opened into the common hepatic bile duct (Fig. 1). The right and left intrahepatic ducts were dilated to five times their normal caliber. The common hepatic bile duct contained many "shadows" which were thought to be intraductal calculi. The contrast medium, however, was unable to pass from the common hepatic duct into the choledochus because of some unrecognizable obstruction. Progress cholangiograms, which were taken twenty minutes later, revealed that the Diodrast had passed from the common hepatic duct into a long pendulous gall bladder. Several shadows were seen within its lumen and these were thought to represent gallstones (Fig. 2). The fact that none of the Diodrast had entered the choledochus,

Fig. 2.—This cholangiogram was taken twenty minutes after Fig. 1. Some of the Diodrast which had passed from the hepatic ducts into the accessory gall bladder clearly outlines the incarcerated stones. The calculi in the common hepatic duct are clearly visualized. Again, the common bile duct is not seen and none of the Diodrast was able to flow into the duodenum. Diagnosis was external biliary fistula, accessory gall bladder containing stones, calculi within the common hepatic bile duct, and complete obstruction of the choledochus.

even at this late period, substantiated the diagnosis of an obstructive lesion. The cholangiograms were interpreted as showing an accessory gall bladder, in addition to stones in the common hepatic bile duct, and an external biliary fistula secondary to a complete obstruction of the choledochus.

The patient was readmitted to the hospital on March 22, 1947, for surgical exploration. The external biliary fistula was traced to the lower end of the common hepatic bile duct, where by traction it had produced a kinking obstruction of the proximal end of the choledochus. The lumen of the collapsed common bile duct could not be identified because of profuse bleeding incident to the severe inflammatory process.

The fistulous tract was excised and ligated at a point where it communicated with the anterior surface of the common hepatic duct. The accessory gall bladder was mobilized and its cystic duct was found to pass upward, going posterior to external fistula, and uniting with the common hepatic duct about one inch above the point where the cystic duct of the main gall bladder had effected its union. The gall bladder was indurated and fibrotic and contained several irregularly shaped stones (Fig. 3, *A* and *B*).

At this point the patient's condition became so precarious that operation had to be terminated, without further exploration. The calculi were removed from the gall bladder and a cholecystoduodenostomy was performed (Fig. 3, *C*). It was hoped that this temporizing procedure would divert the bile into the intestinal tract to facilitate the digestion and assimilation of foods. Even though the choledochus had not been identified, and the common hepatic bile duct was known to contain several stones, no further exploration was warranted. Corrective operations, it was hoped, could be employed later, when the patient's condition improved.

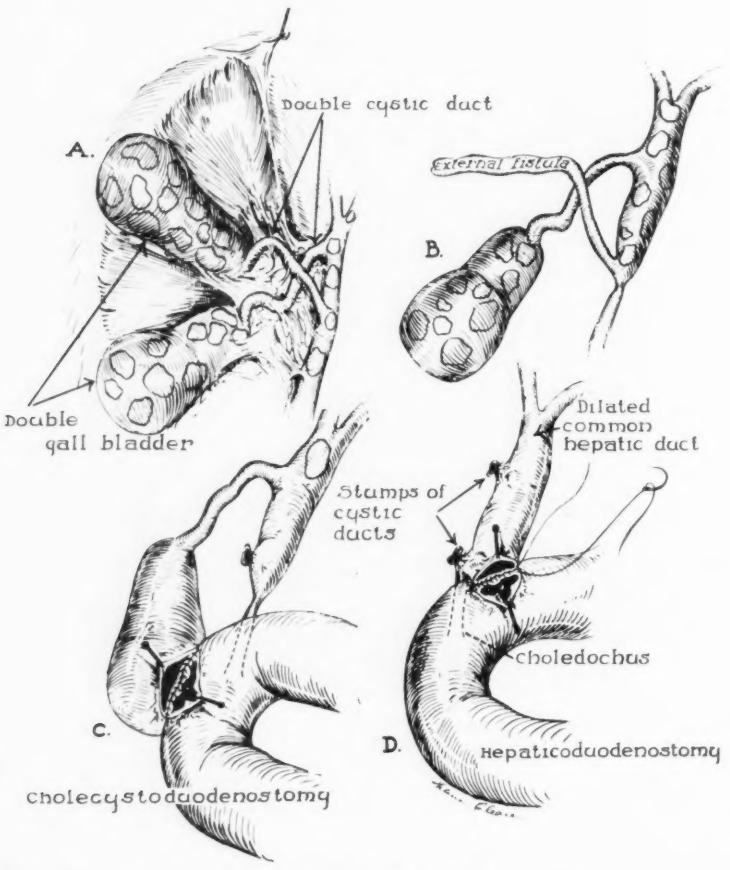


Fig. 3.—*A*, This schematic drawing shows the "double" gall bladders, their individual cystic ducts, and the manner in which they unite with the common hepatic duct.

B, The conditions encountered at the second operation are depicted. The main gall bladder has been removed. The persisting external biliary fistula originated at the point where the cystic duct united with the common hepatic duct, and resulting scar tissue is the obstructed choledochus. Inflammatory reactions and traction of the external biliary fistula have produced a pressure and kinking obstruction of the choledochus.

C, At the second operation the stones were extracted from the gall bladder, the external biliary fistula was excised, and a cholecystoduodenostomy was performed.

D, At the third operation the accessory gall bladder and its cystic duct were removed. The common hepatic duct was divided, the intraductal stones were removed, and a hepaticoduodenostomy was performed.

A very stormy convalescence was experienced despite the repeated transfusion of whole blood and the administration of solutions containing glucose, protein hydrolysates, and penicillin. On the fourth postoperative day the septic temperature ranged from 104.8° to 99.4° F. Blood cultures furnished an abundant growth of gram-positive diplococci and nonhemolytic streptococci. These findings were confirmed on several occasions. The patient was given 250 mg. of streptomycin every four hours, and 100,000 units of penicillin every three hours. When she had received a total of 9 Gm. of streptomycin and 12,800,000 units of penicillin, the blood cultures were reported sterile. During her last week in the hospital the temperature did not rise above 99° F. She was eating well, the jaundice had disappeared, and stools contained normal amounts of bile pigments.

The surgeon who performed the third operation kindly supplied us with the following information. The gall bladder was found to be collapsed and anastomosed to the jejunum. The anastomotic opening was patent and functioning, but no bile was found in the gall bladder indicating an obstruction of the cystic duct. Further exploration indicated that a large calculus within the common hepatice duct had become lodged in the orifice of the cystic duct and had prevented the bile from entering the gall bladder. The obstructing calculus had completely negated the benefits which should have resulted from the cholecystojejunostomy. The stones were removed from the common hepatic bile duct and the gall bladder and cystic duct were excised. As the choledochus could not be identified, the common hepatic duct was anastomosed to the jejunum (Fig. 3, *D*). The patient made an excellent recovery.

DOUBLE GALL BLADDER WITH "Y" CYSTIC DUCTS

CASE 2.—A physician, 50 years old, came to the hospital after having diagnosed his own case as one of "biliary colic." During the past two years he had suffered from recurring attacks of gallstone colic, which required the use of morphine sulfate for relief. Four cholecystographic examinations were made by different radiologists and in no instance was the gall bladder visualized, but several opaque calculi were seen. The paroxysms had increased in both frequency and severity so that he desired to have the offending gall bladder removed.

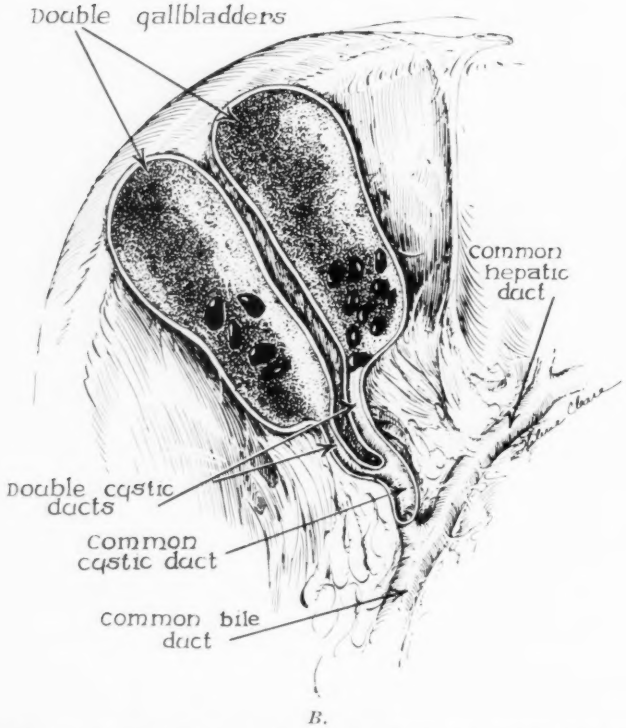
When the ventral surface of the liver was exposed, a large, tense mass was found in the gall bladder fossa. Considerable dissection was required to free the distended gall bladder from the adherent omentum. No difficulties were experienced in removing the gall bladder, nor were any anatomic aberrations of the extrahepatic biliary system detected.

The pathologist submitted the following report. The specimen consisted of a large distended gall bladder, surrounded by considerable amount of adipose tissue. On transecting the gall bladder it was found to contain mucoid material and ten faceted stones, one of which was embedded in the orifice of the cystic duct. Strangely, the entire mass did not collapse when the main gall bladder was open as a tense, ovoid tumefaction was found adherent to the inferior wall. On incising this tumorous mass it was found to be an accessory gall bladder, having no communication with its mate. The accessory organ contained five faceted stones which were floating in thick inspissated bile (Fig. 4, *A*). Each gall bladder had its own individual cystic duct which measured 2 cm. in length. These paired cystic ducts then united to form a common cystic duct which had a length of 1.5 cm. (Fig. 4, *B*). Microscopic studies demonstrated the mucosa to be somewhat hyperplastic and fairly well preserved. There was considerable round-cell infiltration in the walls of both gall bladders. The calculi were of the "mixed type" and were deeply pigmented. Diagnosis was double gall bladders having separate cystic ducts which united to form a single common cystic duct.

DOUBLE GALL BLADDERS RECOGNIZED BY CHOLECYSTOGRAMS

CASE 3.—A merchant, 51 years of age, had been annoyed by pains in the right upper quadrant which were aggravated by severe gaseous eructations. A cholecystographic examination was performed on April 1, 1945. The gall bladder was found to be filled with cholecystographic dye. Its size, shape, and conformation appeared to be normal. No calculi were observed. Just mesial to the normally placed gall bladder was a second shadow,

A.



B.

Fig. 4, *A* and *B*.—The photograph and drawing show the double gall bladders and their cystic ducts. In this instance the two paired cystic ducts fuse to form a common cystic duct before it unites with the common hepatic bile duct.

having a smooth contour and a globoid shape, which was thought to be an accessory gall bladder. Following the ingestion of the fat containing meal both of these shadows were much smaller, but they still retained large amounts of the cholecystographic dye. It was apparent that this patient had double gall bladders, both of which had good powers of concentrating the ingested dyes, but there was a definite disturbance in the motility and emptying functions (Fig. 5). Unfortunately, the patient's digestive disturbances were so few that he refused operative intervention, hence the diagnosis could not be confirmed.

CASE 4.—A physician, 42 years old, had complained of vague distress in the epigastric region. A cholecystogram was done on Feb. 2, 1939, and was interpreted as follows. In the upper right quadrant of the abdomen there were two gall bladders, each of which was filled with the cholecystographic dye. The main gall bladder was suspended from the ventral surface of the right lobe of the liver, so that its long axis was in a vertical plane. The accessory gall bladder, however, occupied a position directly under the twelfth rib and hence lay in an oblique position. There were no changes in the size, shape, or density of their respective shadows following the ingestion of the fat meal. No gall stones were detected (Fig. 6). Apparently the duplicative gall bladders possess normal powers of concentration but exhibit definite interference with motility. Diagnosis was double gall bladders having impaired motor function. Again, the patient refused to submit to surgical intervention so confirmation was impossible.

DISCUSSION

"Double" or "duplex" gall bladders initiate no characterizing clinical syndrome which permits differentiation from other cholecystopathes. Occasionally the radiologist might detect two distinct gall bladder shadows,[4] or note two distinct rows of gallstones,[3] which would indicate the presence of these duplicative abnormalities. The literature contains the report of forty-eight cases of double gall bladders which have been recognized by routine roentgenologic examinations, and in fully 95 per cent the gall bladders still retain sufficient function to concentrate the ingested dyes.

It must be remembered, however, that cholecystography is not infallible, and may invite a false appraisal of the patient's condition. It is not unreasonable to assume that one of the "paired" gall bladders might be so diseased that all function would be lost, while its twin might still retain its normal physiologic properties. Under such circumstances the unaffected organ would present a normal cholecystographic response, while the diseased gall bladder would not be visualized. The radiologist, being unaware of the nonvisualized, pathologic gall bladder, would submit a report based on the visualization of the normal accessory organ, which might read, a "well visualized gall bladder possessing normal functions of concentration and emptying." In view of this report the surgeon might not have the temerity to explore the abdomen for the diseased gall bladder when the roentgenologic examination indicated a "normal functioning gall bladder."

Since the advent of cholecystography, various malformations in the shape and contour of the gall bladder have been reported with increasing frequency. Numerous descriptive terms, such as, bilobed, bifid, biocular, hourglass, dumbbell-like, S shaped (Fig. 7, *B*), and phyrgian-capped, have been used by the radiologist to denote the characterizing deformities. In spite of the fact that these gall bladders have but one cavity, they may produce two distinct gall

Fig. 5.—The cholecystogram outlines two distinct gall bladder shadows. The more laterally placed gall bladder possesses a normal concentrating function, while the accessory and the more mesially placed gall bladder presents a lighter silhouette. There was very little change in these pictures following the ingestion of the fat containing meal. Diagnosis was double gall bladders.

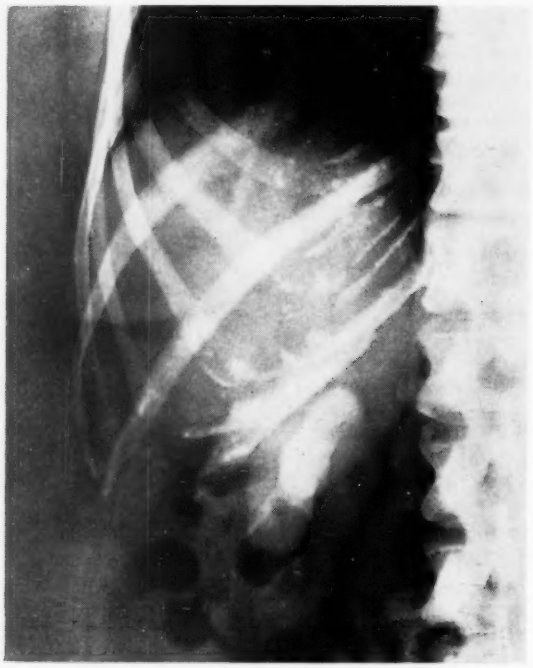
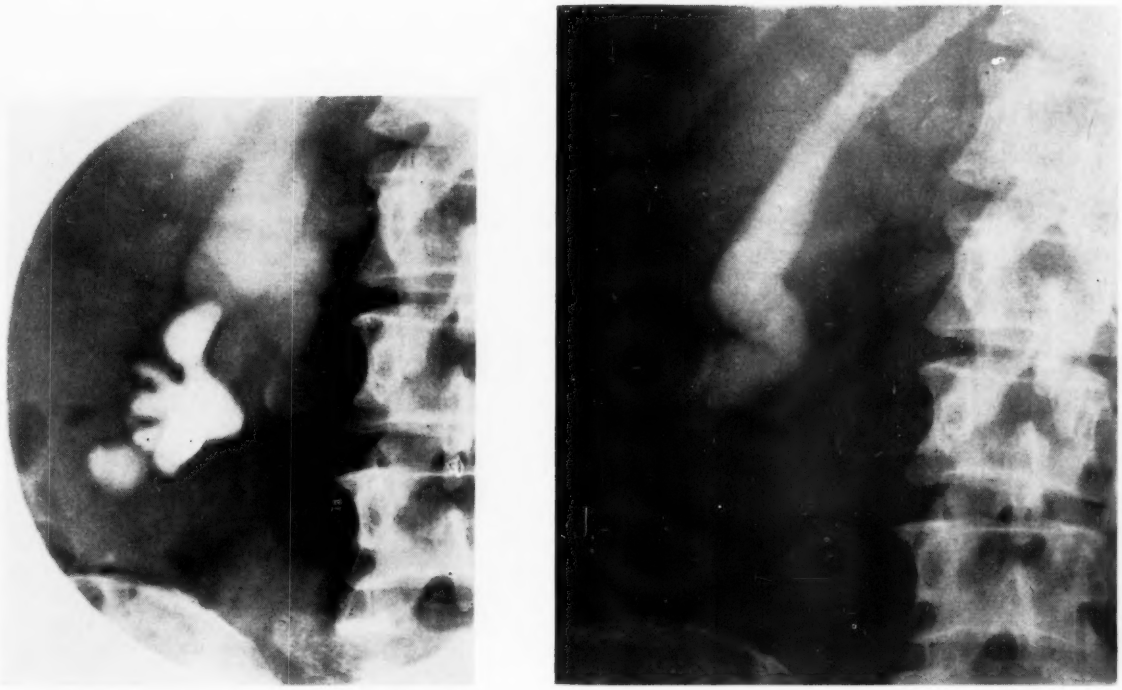


Fig. 6.—Two gall bladders are visualized by the cholecystographic dye. One gall bladder is suspended from the ventral surface of the liver so that it hangs in a vertical plane, while the more mesially placed gall bladder lies in an oblique direction just below the lower level of the twelfth rib. The fact that both gall bladders were visualized indicates practically no impairment in their function of concentration, but there was no alteration in size, shape, or density following the fat meal, suggesting a disturbance of motility.

bladder shadows, if viewed from the anteroposterior position. Allen maintains that if the roentgenograms are taken from several views, such as the anteroposterior, prone, lateral, oblique, and erect positions, that differentiation is possible (Fig. 7). Double gall bladders will present two distinct shadows in all of these positions, while those having malformations of contour only will exhibit a single shadow in some of these various views.



A. *B.*

Fig. 7.—*A*, The cholecystogram shows two silhouettes in the gall bladder area and a large staghorn calculus in the right kidney.

B, Following the removal of the renal calculus a second cholecystogram was taken while the patient was in an oblique position. The gall bladder now appears as a long, redundant structure which was "folded" on itself, which accounted for the two shadows which were seen in the original cholecystogram.

Recognition of congenital anomalies of the gall bladder, whether duplicative or merely changes in contour, can be accurately accomplished by cholangiographic studies while the patient is on the operating table. If the contrast medium, such as Diodrast, is injected into the presenting gall bladder, it will flow into all other segments of the extrahepatic biliary, and the resulting cholangiogram will provide an exact anatomic pattern of existing anomalies. The surgeon can then institute the proper remedial operations. Had such a plan been carried out in our first case, the accessory gall bladder and the intraductal stones would not have been overlooked.

SUMMARY

1. A true "double" gall bladder is a congenital anomaly in which there are two complete gall bladder walls, two separate, noncommunicating cavities, each

of which is drained by its own cystic duct. The paired cystic ducts may empty into the same or different segments of the extrahepatic biliary system, or the separate cystic ducts may fuse in a Y like pattern to form a common cystic duct, which then unites with one of the large biliary radicals. Representative cases are discussed.

2. The bilobed and bifid gall bladders should not be confused with the duplications of the vesica fellea. Anatomic and radiologic differentiations are discussed.

3. Double gall bladders produce no characterizing clinical syndromes which permit differentiation from other cholecystopathies. The roentgenographic identification of double gall bladders has been made by observing two separate rows of gallstones, or by noting two distinct silhouettes of the gall bladder during routine cholecystographic examinations. In two of our cases the paired gall bladders still retained sufficient function so that they were able to concentrate the cholecystographic dye, and hence cast recognizable shadows. In the other two cases, however, the gall bladders were so diseased that roentgenographic visualization was impossible.

4. Cholangiographic visualization of the extrahepatic biliary system, when the abdomen is opened, affords the best method of recognizing any existing anatomic aberrations.

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DUODENAL DIVERTICULITIS WITH PERFORATION

Report of a Case

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In a review of the American and English literature the report of only five cases* of diverticulitis of the duodenum with perforation have been found. This number of cases appears unusually small since diverticula of the duodenum are found in one to two per cent[4, 5] of all gastro-intestinal x-ray series and in three and three-tenths per cent to fourteen and one-half per cent of post mortem[6] and anatomical[7] studies carried out for this particular abnormality.

ETIOLOGY

The reasons advanced for the infrequent occurrence of perforation of duodenal diverticula are (1) comparative sterility and fluidity of the content, (2) the retroperitoneal situation which permits ready distention, and (3) wide-mouthed ostia and inverted supported position (when on the concave side) facilitating dependent drainage.[8] The third explanation lacks substantiation when it is remembered that the ostium is often concealed among the plicae circulares of the duodenum and that little or no muscle is present in the sac wall. Because of these latter conditions retention, which favors bacterial growth and inflammatory involvement, should occur. Weintraub and Tuggle[9] in their work on diverticula of the duodenum demonstrated that retention does occur. Out of 310 patients with duodenal diverticula receiving a barium meal, 52 showed a retention of six or more hours.

SYMPTOMS

The symptoms of perforation of duodenal diverticula are pain, nausea, emesis, and localized tenderness with associated muscular rigidity, fever, and leucocytosis. Shock may or may not be present. The severity of the symptoms will depend upon the size and rapidity of the perforation and the direct or indirect communication with the general peritoneal cavity. In all the reported cases except two, an incorrect diagnosis of perforated peptic ulcer, perforated appendicitis, or acute cholecystitis was made. The diagnosis was made by roentgen studies in the case reported by Lucinian.[10] The diagnosis of a diverticulum of the duodenum was made in the case reported by McQuay,[11] but the perforation was not recognized until operation. The two latter cases were treated as elective surgical cases.

PATHOLOGY

The diverticulum may vary in size from a few millimeters to 8 to 10 cm. in diameter. It is usually found on the inner and posterior surface of the duo-

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*The cases presented by Huddy and Costello have been reported as duodenal diverticulitis with perforation. On review of their original papers, no mention of perforation was made.[1, 2] The case of Erkes, quoted by Brown and McHardy, was originally reported in the German literature.[3]

denum along the line of entrance of the vessels. The most common site is the second portion of the duodenum. The first part of the duodenum is the second most common site and the third part is the least common location. The diverticulum is usually dorsal to the head of the pancreas, but may be anterior, between the pancreas and the duodenum, or even within the head of the pancreas. If the diverticulum arises from the posterior part of the duodenum the serosal coat will be absent. The ostia vary in size, usually wide in the smaller diverticula and rather narrow in the larger ones. The mucosa is a continuation of the duodenal mucosa. Pancreatic rests may be found in the walls. There may or may not be a muscular layer and if one is present it is usually thin. Some authorities believe that the congenital diverticulum does not have a muscular layer, while the acquired diverticulum does. The inflammatory reaction seen in diverticula is analogous to that observed in the appendix. It may vary from congestion with leucocytic infiltration to suppuration and gangrene with or without perforation.[12]

CASE REPORT

History.—The patient, a white man, 71 years of age, complained of generalized abdominal pain on admission. About 10 A.M., Oct. 18, 1947, he was stricken with sudden sharp pain in the epigastric region, so excruciating that he collapsed. He was found a few minutes later on the floor grimacing in acute pain and lying on the right side with thighs acutely flexed on the abdomen, afraid to move for fear of increasing the pain. He was removed to a hospital where he was placed in high Fowler's position with oxygen tent (presumably a coronary thrombosis was suspected). He felt nauseated but did not vomit. The pain spread to the right upper quadrant but did not penetrate to the back or to either shoulder. It persisted without let-up. At twelve noon he received a quarter of a grain of morphine sulfate. The dose of morphine sulfate was repeated at 3:30 P.M.

The patient arrived at the University Hospital at 4:30 P.M. In spite of the morphine sulfate received, he was still in acute distress. He gave no history of similar attacks. He had, however, on several occasions had an "inside swelling" which was relieved by belching. He had had episodes of melena which lasted for one or two bowel movements and then cleared.

Physical Examination.—Temperature was 98.6° F., respirations were 22 per minute, and blood pressure was 170/80. He was a well-preserved, white man in acute distress. There was no evidence of shock. He lay on the right side with the thighs flexed acutely on the trunk and moved with reluctancy into the recumbent position when asked. The examination of the heart and lungs was negative. The abdomen was moderately distended. Tenderness was present over the entire abdomen but was more pronounced in the upper right quadrant. Generalized tenseness of the abdomen was present with boardlike rigidity of the upper right rectus muscle. No peristalsis was audible and no mass was palpable. Rectal examination was negative except for a grade 3 prostatic enlargement.

Operation.—The patient was taken to the operating room at 6 P.M., eight hours after the onset of pain. Under cyclopropane anesthesia, administered through an endotracheal tube, and supplemented with curare, the abdomen was opened through a right upper rectus muscle-splitting incision. Examination of the abdominal cavity disclosed a small amount of yellow fluid. The intestines were quiet and there was no serosal exudate. Just lateral to and beneath the duodenohepatic ligament was a bulge of the retroperitoneum. On reflection of the fixed portion of the first and second part of the duodenum by dividing the parietal peritoneum along the lateral border, a bloody, bile-containing fluid escaped into the peritoneal cavity. After suction cleared the operating field, a duodenal diverticulum was seen extending dorsal to the head of the pancreas superiorly and medially. The serosal surface was covered by a fibrinous exudate. A perforation about 2 cm. in diameter was present at

the apex. The diverticulum was 7 cm. in length, adherent to the surrounding tissues, and attached by a stalk near the ampulla of Vater. The communication into the duodenum was large enough to admit easily the index finger. A partial excision of the diverticulum with plication of the remaining portion was decided upon because of the size of the opening into the duodenum and its relation to the ampulla of Vater. Over four-fifths of the diverticulum was excised. The remaining portion was plicated transversely to the axis of the duodenum with a continuous suture of zero chromic catgut. This was re-enforced by a continuous Lembert suture of zero chromic catgut. A fat pad of omentum was then sutured over this area for further protection. The parietal peritoneum was closed along the second portion of the duodenum. The peritoneum along the first portion and the angle of the duodenum was left open. Drainage through stab wounds in the anterior abdominal wall was decided upon. Penrose drains were placed into the lesser omental cavity, the subphrenic space, the right gutter, and the pelvis. The abdominal wall was closed with zero chromic catgut, skin silk, and braided silk stay sutures. The patient received two pints of whole blood during the operation and was in good condition when taken from the operating room.

Postoperative Course.—The postoperative course was complicated by acute urinary retention and a wound infection. The urinary retention was relieved by placing an indwelling No. 20 Foley catheter into the bladder. The wound infection responded rapidly to drainage and daily irrigations with sodium bicarbonate (the pH of the wound was acid).

Pathology Report.—The specimen consisted of a piece of tissue measuring 6 cm. in length. The inner surface was smooth. It represented mucosa of the intestine. The serosal surface was necrotic and covered by a fibrinous exudate.

On section the wall was seen to consist of mucous, submucous, muscular, and serosal coats. All coats showed an inflammatory exudate.

ANALYSIS OF REPORTED CASES (TABLE I)

Age.—In this group of cases the youngest patient was 36 years of age and the oldest was 71, with an average of 57.2 years. The age incidence is not sur-

TABLE I. ANALYSIS OF CASES OF PERFORATION

AUTHOR	AGE (YR.)	SEX	PREOP. DIAGNOSIS	LOCATION OF DIVERTICULUM		PERFORATION	BASE	RESULTS
				DUODENUM	PANCREAS			
Monsarrat[13]	58	F	Acute cholecystitis	2nd portion, concave side	Dorsal	Apex	Stalk	Recovery
Lucinian[10]	36	F	Duodenal diverticulitis with perforation into pancreas	Concave side near ampulla of V.	In pancreas	Apex	Stalk	Recovery
McQuay[11]	65	M	Duodenal diverticulitis	2nd portion, concave side	Into pancreas	No comment	Stalk	Recovery
Beaver[14]	54	M	Perforated appendix	Concave side near ampulla of V.	Dorsal	Apex	No comment	Died
Boland[15]	59	M	Perforated ulcer	Concave side near ampulla of V.	Between duodenum and head of pancreas	Apex	Stalk	Died
Present case	71	M	Perforated ulcer, posterior	Concave side near ampulla of V.	Dorsal	Apex	Stalk	Recovery

prising when it is remembered that 5 per cent[16] of the people over 55 years of age having a gastrointestinal x-ray series are found to have one or more diverticula of the duodenum, while in the over-all age group only 1 to 2 per cent[16] have diverticula. The chance of perforation then is four to five times greater in the age group over 55.

Sex.—There were four men and two women in this group. The series is too small to draw any conclusions as to the frequency of occurrence in the two sexes.

Preoperative Diagnosis.—An incorrect diagnosis was made in all cases in which an immediate operation was necessary (see Table I). The remaining two cases did not require an immediate operation, so a thorough diagnostic study, including roentgen studies, was possible. In the case reported by Lucinian,[10] the correct diagnosis was made with a gastrointestinal x-ray study. In McQuay's case[11] the diagnosis of diverticulum of the duodenum was made, but the perforation into the head of the pancreas was not demonstrated until the abdomen was opened.

Location of the Diverticulum.—In all the cases the diverticulum arose from the concave side and the second portion of the duodenum. This is in conformity with the findings of Ackerman.[17] He stated that 95 per cent of all diverticula of the duodenum arise from the concave side and 75 per cent of these from the second portion of the duodenum. Of the 75 per cent arising from the second portion, the great majority occur within 2½ cm. of the ampulla of Vater. The occurrence in the second portion of all the cases of perforation must be based on the frequency of diverticula in this location, for no great anatomic or physiologic difference exists between the diverticula of the second portion and those of other parts of the duodenum.

Perforation and Base of the Diverticulum.—The diverticulum arose by a stalk and perforated at its apex in all the cases in which there were data on these two points (Table I). Since all the cases commented upon arose by a stalk and presented an opening into the duodenum, smaller than the diameter of the diverticulum, it is thought that the significance of this existing anatomic condition must be considered as a causative factor. Retention would certainly be more likely and pressure from within the diverticulum greater.

The frequency of perforation of the diverticulum at the apex is best explained by the damaged blood supply. The vascular system supplies the diverticulum through its attachment to the duodenum. Ischemia and necrosis, which precede perforation, would result in the more distal part first.

RESULTS

In all the cases where the diverticulum was found and treated, recovery resulted. In the two cases where the lesions could not be demonstrated at operation and treatment consisted of drainage without closure of the perforation, death resulted. The mortality for all the cases was 33⅓ per cent. In the cases in which immediate surgery was necessary (four in number), the mortality was 50 per cent.

SUMMARY

1. A review of the literature of diverticulitis of the duodenum with perforation has been presented.

2. Another case of diverticulitis of the duodenum with perforation has been added.

3. Perforation of duodenal diverticula and peptic ulcers present the same clinical symptoms.

4. In all cases the diverticulum arose by a stalk and the perforation occurred at the apex.

5. The mortality for all the cases was 33⅓ per cent while the mortality for the acute cases requiring prompt surgery was 50 per cent.

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THE EFFECT OF SURGICAL DEVASCULARIZATION OF THE STOMACH ON THE PRODUCTION OF MANN-WILLIAMSON ULCERS

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DEVASCULARIZATION of the stomach by ligation of the major portion of the arterial supply of the organ has been proposed as a method of treatment for chronic duodenal ulcer.

Somervell[1] reported the use of the procedure with or without complementary gastrojejunostomy in over 400 cases in Travancore, South India. He suggested that interrupting the major portion of the arterial supply to the stomach results in a "physiologic gastrectomy," with ischemic atrophy and diminished function of the chief and parietal cells of the gastric mucosa. He compared the clinical results obtained in this series with those obtained in comparable series of gastric resections and of simple posterior gastrojejunostomies for duodenal ulcer. His end results after a follow-up period of from one to five years indicated that in the physiologic gastrectomy group there was a more persistent reduction in gastric hyperacidity, a lower mortality rate, and a lower incidence of recurrent ulceration than in either of the groups with gastric resection or gastroenterostomy.

Any method proposed for the management of peptic ulcer can be evaluated in part by determining its effect on the production of experimental ulcers in animals.

LeVeen[2] has noted that the typical peptic ulcers in the first portion of the duodenum, ordinarily induced by intramuscular injection of histamine-in-beeswax, did not occur in animals who had undergone devascularization operations. With extensive ligation, however, vascular ulcers occurred on the mid-portion of the greater curvature of the stomach. Baronofsky,[3] however, in a similar study found that ligation of three of the large arteries of the stomach and additional small arteries coming from the gastroepiploic vessels failed to prevent histamine-provoked ulcer in his series of five dogs.

The Mann-Williamson procedure[4] has been used in many experimental laboratories as a method of evaluating peptic ulcer therapy. In this operation the alkaline juices of the duodenum are shunted into the terminal ileum. Typical peptic ulcers occur in a large percentage of these experiments as a result of the un-neutralized hydrochloric acid and pepsin, acting upon the jejunal mucosa. In the original series of Mann and Williamson fourteen of the sixteen animals developed these experimental ulcers. More recent workers report the occurrence of experimental ulcers following the Mann-Williamson procedure in over 95 per cent of operated animals.[5, 6] In our experimental laboratory,

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this procedure has been used to evaluate several proposed methods of ulcer therapy.[7, 8] In this study we have attempted to determine the effect of the procedure of "physiologic gastrectomy" on the production of Mann-Williamson ulcers.

METHODS

Six normal mongrel dogs were used in this study. Under intravenous pentothal sodium anesthesia, using aseptic technique, all the arteries on the lesser curvature and approximately nine out of ten of the smaller vessels coming from the gastroepiploic vessels on the greater curvature were ligated with 00 silk. This was combined with an immediate Mann-Williamson procedure in two dogs, and in the four other animals the Mann-Williamson operation was done as a second stage, after an interval of time, varying from fourteen to eighty-one days. In these animals, further ligation of several blood vessels to the stomach at the time of the second laparotomy was performed.

The Mann-Williamson procedure was performed as follows: the stomach was divided at the pylorus, and the duodenal end was inverted and closed with three layers. The jejunum was severed three or four inches distal to the ligament of Treitz, and the proximal end was joined to the terminal ileum about twelve inches from the ileocecal junction by end-to-side anastomosis. The distal end of the severed jejunum was joined to the pylorus by an end-to-end anastomosis.

In five control animals in which the Mann-Williamson procedure was done without gastric devascularization, ulcers occurred in all cases. The average duration of life from the time of the Mann-Williamson operation to the time of death from ulcer in the control series was twenty-two days.

EXPERIMENTAL RESULTS

All the dogs which survived over one week developed typical peptic ulcers of the jejunum. In the two animals that died from ulcer (Fig. 3) and (Fig. 4) the survival time was 95 and 111 days after the Mann-Williamson operation (Table I). The other two dogs that died from other causes had definite jejunal ulcers (Figs. 1 and 2).

COMMENT

It will be noted in Table I that in spite of the extensive devascularization of the stomach the four animals which survived longer than six days all developed jejunal ulcers. These ulcers were identical with ulcers seen in the control animals.

TABLE I. THE RESULTS OF SUBTOTAL GASTRIC DEVASCULARIZATION FOLLOWED BY THE MANN-WILLIAMSON OPERATION IN DOGS

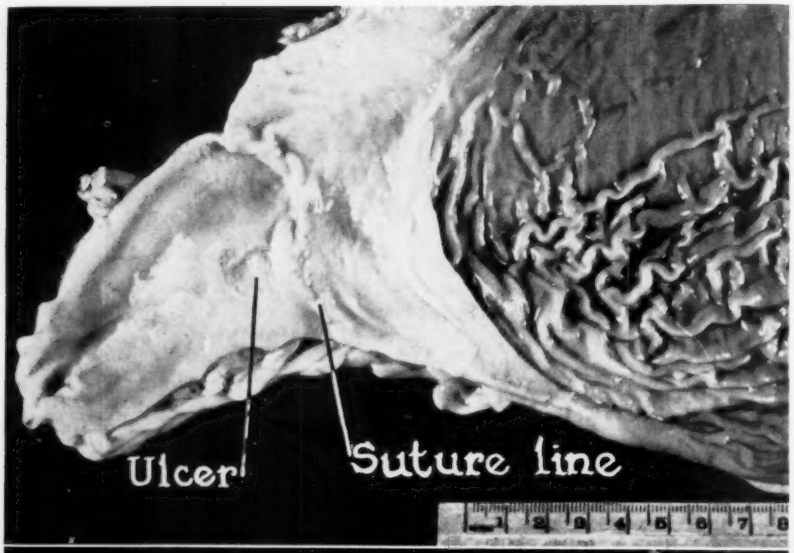
DOG	DATE	OPERATION	SURVIVAL AFTER M-W* (DAYS)	AUTOPSY FINDINGS
1	2/13/47 2/29/47	Devascularization M-W*	2	Atelectasis, no ulcer
2	3/28/47	Devascularization M-W	6	Subhepatic abscess, no ulcer
3	2/ 8/47 2/21/47	Devascularization M-W	33	Gastric trichobezoar, jejunal ulcer
4	1/30/47 2/21/47	Devascularization M-W	44	Chronic, partial intestinal obstruction; jejunal ulcer
5	3/21/47 6/13/47	Devascularization M-W	95	Jejunal ulcer with perforation
6	4/22/47	Devascularization M-W	111	Jejunal ulcer with perforation

*Mann-Williamson.

The length of time of survival in the two experimental animals that died from the effects of Mann-Williamson ulcer was 95 and 111 days, while our control animals survived an average of only 22 days before death from ulcer. In such a small series little significance can be attached to this because other workers have reported survivals in control animals up to three and four months.

Inasmuch as the Mann-Williamson ulcer is the result of the action of unneutralized gastric acid and peptic secretion, any procedure which might be expected to prevent these ulcers should produce a marked reduction in the

Fig. 1.



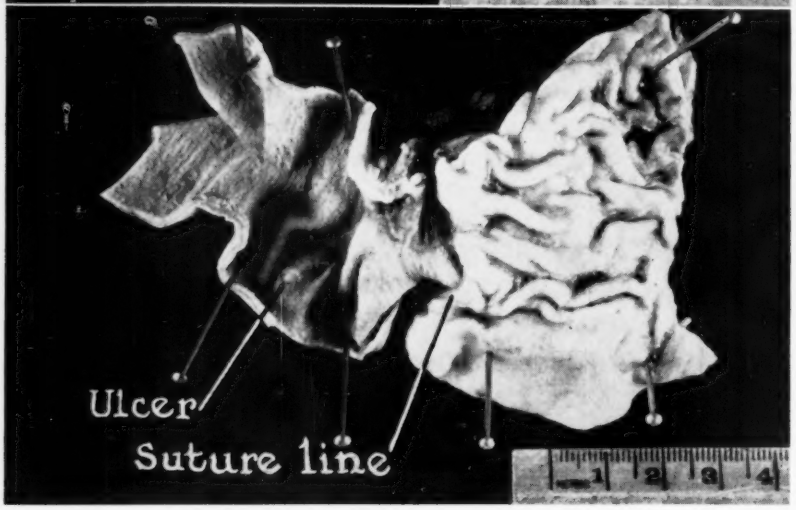


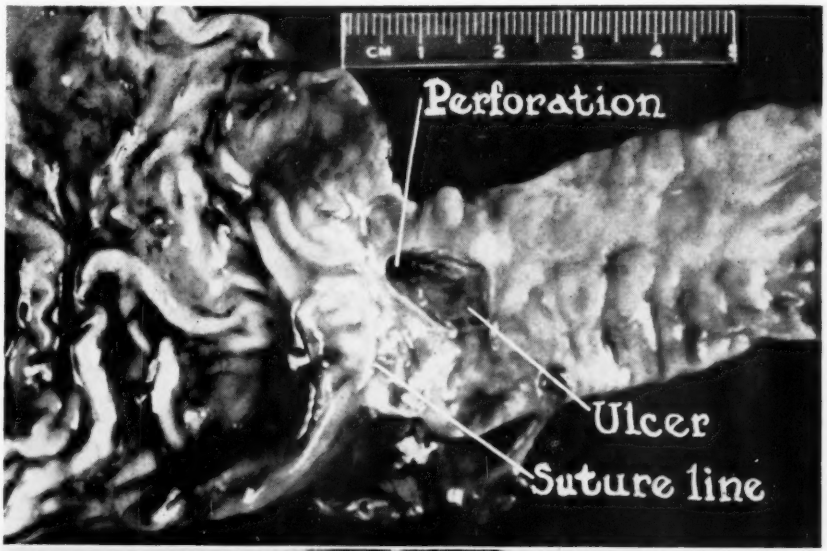
Fig. 2.

Fig. 1 (Dog 3).—Forty-six days after devascularization and thirty-three days after Mann-Williamson operation and further devascularization; death from trichobezoar and malnutrition. Note jejunal ulcer just beyond anastomosis.

Fig. 2 (Dog 4).—Sixty-six days after devascularization and forty-four days after Mann-Williamson operation and further devascularization; death from chronic partial intestinal obstruction. Note superficial ulcer in jejunum.

amount or concentration of gastric secretion. Layne and Bergh[9] and Bernheim[10] did repeated gastric analyses on dogs at varying intervals after subtotal gastric devascularization and noted no change in gastric acidity. Babkin[11] ligated most of the gastric arteries in three dogs with esophagostomy and gastric metal fistulas. He reported practically no change in free or total acidity, total chloride concentration, or peptic activity following histamine administration or sham feeding of meat. LeVeen[2] noted a reduction in total volume but

Fig. 3.



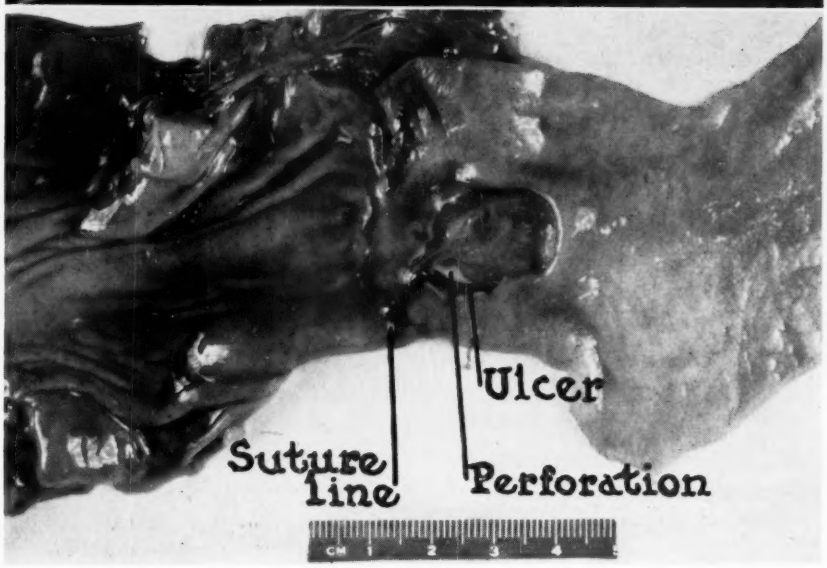


Fig. 4.

Fig. 3 (Dog 5).—One hundred seventy-nine days after devascularization and ninety-five days after Mann-Williamson operation and further devascularization. Death from perforated jejunal ulcer.

Fig. 4 (Dog 6).—One hundred eleven days after devascularization and Mann-Williamson operation. Death from perforated jejunal ulcer.

not of concentration of gastric secretion after surgical interruption of gastric blood flow.

The fact that the formation of Mann-Williamson ulcers was not prevented by arterial ligation of the stomach is also consistent with the findings in a complementary study in our laboratory[12] that histologic changes in the gastric mucosa are minimal and that no significant decrease in gastric acidity is noted following this procedure.

No evidence of vascular ulcers or massive gangrene of the greater curvature, which were noted by other observers,[2, 9] was found in our animals. There was no disturbance in healing of the anastomosis at the pylorus, even after almost complete devascularization. The remarkable ability of the stomach to withstand interruption of its arterial blood supply is probably due to the existence of a vascular network which supplies all parts of the organ when one small vessel to the stomach is left intact. Revascularization from adhesions to surrounding organs occurred to a slight extent.

CONCLUSIONS

Subtotal devascularization of the stomach failed to protect dogs from Mann-Williamson ulcers in our series.

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A METHOD FOR PRODUCING COARCTATION OF THE THORACIC AORTA IN DOGS

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IN ORDER to study the physiologic effects of partial occulsion of the thoracic aorta in dogs, it became necessary to devise a method that would reduce the lumen, yet, would not result in thrombosis or rupture of the vessel. In the past, many attempts to occlude partially and completely the aorta[1, 2, 3] have been carried out. The procedures directed toward the external compression of the vessel have been almost uniformly unsuccessful because of necrosis of the wall of the aorta. Owings,[4] however, was successful in eventually ligating the aorta in 6 out of 75 dogs, using rubber bands applied at multiple operations.

Pearse[3] tried many methods of partial occlusion of the aorta. He used intravascular springs and external bands of cellophane to promote scarring. Blalock and Park,[5] in devising an operation for coarctation of the aorta, reduced the flow through the thoracic aorta by complete ligation of the vessel and then anastomosing the left subclavian to the distal aorta. This procedure did not result in any remarkable change in the blood pressure in upper and lower extremities.

It was evident, then, that for a method of partial aortic occlusion to be successful, it would have to avoid compression of the pulsating aorta, yet not produce thrombosis, as dogs do not survive sudden aortic occlusion. It seemed that some method of intravascular occlusion would be likely to succeed. Hufnagel[6] showed that segments of the thoracic aorta could be replaced with Lucite tubes with survival of the animal for long intervals. Lucite or polymerized methacrylate is a nonwettable plastic that produces little tissue reaction and can be easily machined to desired specifications. The first method attempted was with a Lucite tube lined with a free vein graft.

The Lucite tube was 1 cm long, with varied sized external and internal diameters. The large vein, usually the inferior vena cava, was then pulled through the lumen of the tube and everted over each end in such a way that the intimal surface was exposed to the blood current. The aorta was then incised transversely, and the tube placed in lumen resulting in a stricture of the vessel 1 cm. long, that abruptly decreased the lumen. Both the thoracic and abdominal aorta were used. In only one animal did the vessel remain patent as long as six days. Table I shows the results in this group of animals.

From autopsy studies of the animals, it was apparent that the following events took place. Due to turbulence set up by the blood passing through the long stricture, thrombosis developed at the distal end, and then filled up the tube. In the thoracic aorta this frequently resulted in rupture of the aorta from the pulsations against the thrombosed tube.

In order to prevent turbulence, a Lucite tube was designed as shown in Fig. 1. In this tube the point of greatest constriction was close to the proximal

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TABLE I. DATA ON DOGS WITH PARTIAL OCCLUSION OF THE AORTA FROM VEIN-COVERED LUCITE TUBES

DOG	LOCATION OF STENOSIS	SURVIVAL TIME AFTER OPERATION (DAYS)	CAUSE OF DEATH	CONDITION OF AORTA AT SITE OF STENOSIS	MINUTE THORACIC AORTA OCCLUDED
4	Abdominal aorta	4	Wet gangrene of both lower extremities	Thrombosis at site of stenosis; thrombus extended into trifurcation	
3	Abdominal aorta	19	Killed at end of 19 days	Thrombosis at site of stenosis 3 days after operation; thrombus extended into trifurcation	4
10	Abdominal aorta	30	Killed at end of 30 days	Thrombosis at site of stenosis 5 days after operation; thrombus extended into trifurcation	
6	Thoracic aorta	1	Paralysis of lower one-half of body, infarction of kidneys	Thrombosis at site of stenosis	90
7	Thoracic aorta	6	Hemorrhage	Thrombosis at site of stenosis with erosion of vessel wall	25
8	Thoracic aorta	2	Paralysis of lower one-half of body, infarction of kidneys	Thrombosis at site of stenosis	19
9	Thoracic aorta	6	Hemorrhage	Thrombosis at site of stenosis with erosion of vessel wall	22
11	Thoracic aorta	0	Died 30 min. after aorta, clamps removed	Thrombosis at site of stenosis	32

TABLE II. DATA ON DOGS WITH PARTIAL OCCLUSION OF THE THORACIC AORTA WITH A LUCITE TUBE

DOG	LOCATION OF STENOSIS	SURVIVAL AFTER OPERATION	COMPLICATIONS OR CAUSE OF DEATH	CONDITION OF AORTA AT SITE OF STENOSIS	TIME THORACIC AORTA OCCLUDED (MIN.)
14	Thoracic aorta	Still alive 5 mo. after operation	Slight spasticity of lower extremities	Still patent; arterial pulsations in lower extremities	38
16	Thoracic aorta	Still alive 4 mo. after operation	None	Still patent; arterial pulsations in lower extremities	21
22	Thoracic aorta	Still alive 3 mo. after operation	None	Still patent; arterial pulsations in lower extremities	21
23	Thoracic aorta	7 days	Hemorrhage into left chest	No thrombosis; erosion of aorta at proximal end of tube from an extra ligature applied too tightly	16
24	Thoracic aorta	Killed 5 weeks after operation	Spasticity of lower extremities; severe hemorrhage at time of operation	Lucite tube in place; lumen patent	29

end. The inside bore was "streamlined" both proximal and distal to the narrowest area to minimize turbulence. The tube was 1½ inches long, 5/16 inch outside diameter, and 2/16 inch at the narrowest inside diameter. The total degree of constriction of the aorta by the insertion of this tube varies in different dogs, but in the ones presented here, the diameter of the aorta was reduced about 75 per cent. To prevent the sedimentation of the formed elements of the blood on the tube, all surfaces of the Lucite tube were highly polished.

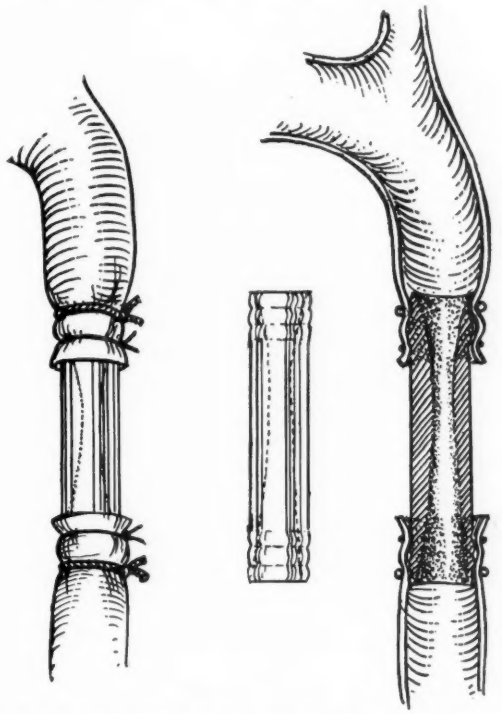

Fig. 1.—Drawing showing a Lucite tube used to replace a segment of the thoracic aorta. The Lucite tube is 1½ inches long, 5/16 inch outside diameter, and 2/16 inch at narrowest inside diameter. The lumen of the tube is constructed so that the turbulence is minimized by streamlining the bore.

The operation on the dogs was carried out with positive pressure anesthesia, and the thorax was entered through the sixth interspace. The thoracic aorta was mobilized by division of the first four pairs of intercostal arteries. Then with two clamps on the aorta, placed 1 inch apart, the vessel was divided, and the Lucite tube was placed in the open ends of the artery as shown in Fig. 1. A ligature of No. 1 silk was tied very tightly and placed next to the cut ends of the vessel. The second pair of ligatures was of heavy braided silk and tied loosely, so that the aorta would be pressed against the Lucite tube just tightly enough to hold, but not to cause necrosis of the vessel wall. This procedure was carried out in five dogs, and the data are shown in Table II.

Dog 23 was the only fatality in the group. Following unusual exercise, seven days after operation, the animal died from rupture of the proximal aorta. It had been necessary to apply a third ligature at the proximal end of the tube because of inaccurate placement of the ligature of braided silk.

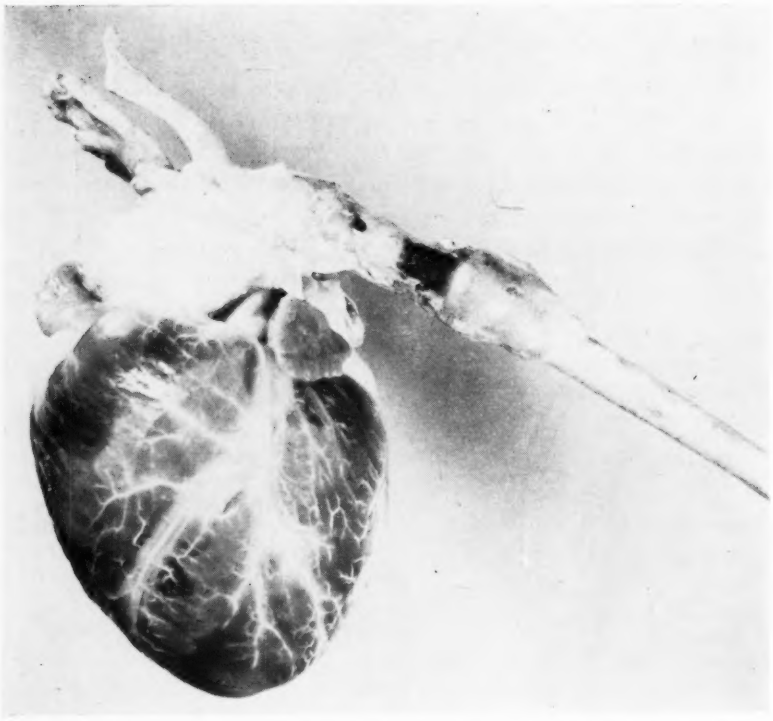

Fig. 2.—The heart and aorta of Dog 24, five weeks after the insertion of a Lucite tube in the descending aorta. The tube is shown as a dark area in the first part of the descending aorta.

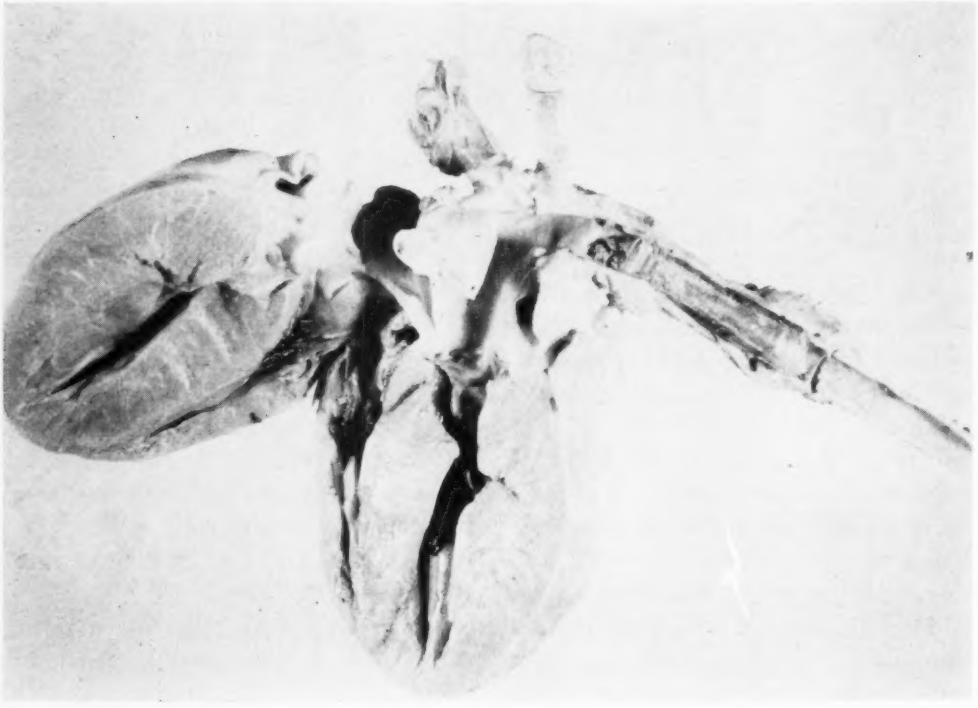

Fig. 3.—The same specimen as in Fig. 2, but with the aorta sectioned longitudinally. The Lucite tube is in place. At upper end of the tube a small thrombus can be seen, however, this does not encroach on the lumen.

Evidence of damage to the spinal cord was present in two of the animals. Dog 1 had the aorta occluded for thirty-eight minutes and developed slight spastic paraplegia, but, after two weeks, only minimal spasticity remained. Dog 6 developed complete spastic paralysis of the lower one-half of the body after occlusion of the aorta for twenty-eight minutes. The prolonged occlusion was complicated at the time of operation by severe hemorrhage due to slipping of the Lucite tube from the end of the proximal aorta. In the remaining three animals in Table II, and the two in Table I that survived longer that twenty-four hours, paralysis did not occur. The maximum time of aortic occlusion in the latter animals was twenty-five minutes.

Table III shows the mean blood pressure changes before and after operation. All measurements were made with a No. 18 needle and recorded by a mercury manometer.

TABLE III. BLOOD PRESSURE CHANGES IN DOGS AFTER PARTIAL OCCLUSION OF THE AORTA

DOG. NO.	PREOPERATIVE MEAN BLOOD PRESSURE		POSTOPERATIVE MEAN BLOOD PRESSURE	
	CAROTID	FEMORAL	CAROTID	FEMORAL
14	Not recorded	110	188	150
22	Not recorded	134	194	134
16	Not recorded	128	204	148
24	110	108	184	144

Pressures recorded under nembutal anaesthesia with a mercury manometer by needle puncture.

Dog 5 was killed at the end of thirty days. Fig. 2 shows the aorta with the Lucite tube in place, while Fig. 3 shows the same specimen after division longitudinally with the Lucite tube still in situ. There is one small area of thrombosis and dilation of the artery just proximal to the Lucite tube that did not encroach on the lumen. The tube was completely encapsulated with a thick layer of fibrous tissue that had fixed the tube in place, and the inside of the tube was smooth without clot or fibrin on its surface.

CONCLUSION

1. A method of stenosis of the thoracic aorta in dogs has been devised that permits prolonged survival of the animals.

2. The stenosis of the thoracic aorta causes mean blood pressure in the carotid artery to increase more than the mean pressure in the femoral artery.

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Case Reports

THE NEED FOR EARLY SURGICAL TREATMENT OF CONGENITAL ANOMALIES OF THE GREAT VESSELS

Case Report—Coarctation of the Aorta, Patent Ductus Arteriosus, and Mycotic Aneurysm

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THE purpose of this report is to direct attention to the group of congenital anomalies of the great vessels which formerly were incurable, and frequently fatal, but which now may be successfully treated by operation. Patent ductus arteriosus and coarctation of the aorta are two of the more common ones belonging in this group.

This is a report of a patient who had both of these anomalies, unrecognized until complicated by a blood stream infection. Although the infection was eradicated by treatment with penicillin, it had already damaged the wall of the aorta. A mycotic aneruysm developed, which bled into a bronchus on several occasions. Surgical treatment was considered at that time, but because the patient was such a poor surgical risk, operation was deferred. The patient died from a massive hemorrhage into the lungs.

This case emphasizes the desirability of early surgical treatment of these conditions before the development of complications which might then make the operation unsuccessful.

CASE REPORT

A 12-year-old white girl was admitted to the hospital because of fever, cough, and chest pain of one week's duration. At the age of 3 years a diagnosis of congenital heart disease had been made on the finding of a cardiac murmur. Except for episodes of unexplained fever and night sweats she experienced no difficulty; her activity was unrestricted.

Admission examination and laboratory studies revealed a clinical picture consistent with the following diagnoses: coarctation of the aorta, patent ductus arteriosus, enlargement of the heart, and bacteremia with emboli. The temperature was 40.5° C., respirations were 30 per minute, the pulse rate was 130 per minute, the blood pressure in the arm 150 mm. Hg systolic, 60 mm. Hg diastolic, and in the leg 90 mm. Hg systolic, 20 mm. Hg diastolic. The heart was enlarged, and a loud rumbling machinery-like murmur was heard over the pulmonic area. The femoral pulse was not perceptible. The liver and spleen were enlarged and tender. The right big toe was swollen, red, tender, and painful. There were many large petechiae on the left foot. X-ray pictures of the chest showed minimal notching of the ribs and an enlarged heart with fullness of the pulmonary conus. The electrocardiogram showed a sinus tachycardia with alternation of QRS complex in Lead I. The hemoglobin content of the blood was 70 per cent, the white blood cell count 12.8 thousands, and the red blood cell count 3.9 millions. Blood cultures were made on several occasions and showed hemolytic *Staphylococcus aureus.*

The clinical course over the next two months showed improvement under treatment with penicillin and other supportive measures. The fever subsided, the blood stream became sterile, and the peripheral infarcts healed. The patient had apparently recovered completely when she suddenly complained of back pain, coughed up bright red blood, became markedly cyanotic, and went into shock. Examination of the chest, clinically and by x-ray, revealed

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atelectasis of the lower and middle lobes of the left lung, with displacement of the mediastinum to the left. The roentgenograms also showed a large shadow in the posterior upper mediastinum, which had not been present on previous examinations. The subsequent clinical course and further radiographic studies showed that the patient had developed a mycotic aneurysm of the aorta, which had ruptured into the left main stem bronchus. Angiocardiograms showed concentration of the dye in the mediastinal mass, absence of dye in the descending aorta, and persistence of dye in the pulmonary conus, corroborating the clinical impression of the triad—mycotic aneurysm, coarctation of the aorta, and patent ductus arteriosus.

The patient was seen in consultation by another doctor, who advised surgical intervention and outlined a program of action. The patient was treated conservatively with sedation, oxygen, and small blood transfusions in the hope that she would recover sufficiently from the acute episode to permit the use of an anesthetic for operation. The operative treatment planned would have required the excision of the aneurysm, and the resection of

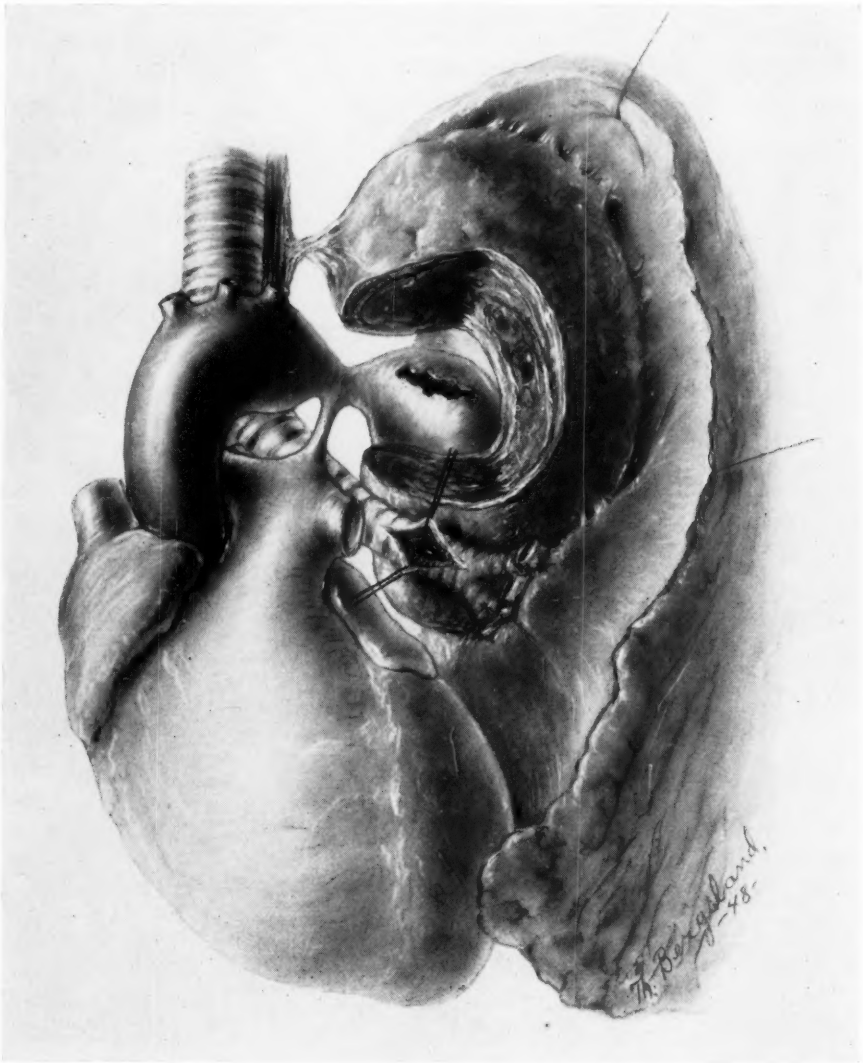

Fig. 1.—Drawing of the specimen showing patent ductus arteriosus, coarctation of the aorta, and the mycotic aneurysm of the aorta which ruptured into the left bronchus. A section of the aneurysm has been removed to show the vascular anomalies.

the narrowed and diseased aorta. In the event that the segment of vessel resected would have been too long to allow for a direct end-to-end anastomosis, two alternatives were considered as methods of closure of the vessel. The carotid or subclavian artery could be transplanted into the lower end of the aorta,[7] or second, a free segment of autogenous vein or artery could be grafted between the open ends of the aorta.[4] Although due consideration was given to the magnitude of the planned procedure, it seemed to be the only recourse if the patient's life were to be saved. Unfortunately, before the patient recovered enough to permit operative intervention, she had three other episodes of hemorrhage, the last of which proved fatal.

The post-mortem examination showed a localized constriction of the aorta which was located at the point of insertion of the patent ductus arteriosus (Fig. 1). Just distal to the coarctation, the anterior wall of the aorta was eroded. The mycotic aneurysm had developed from this point and had extended anteriorly between the left lung and the heart, and inferiorly to the left main stem bronchus, into which it had eroded. Both lungs were filled with blood. The aneurysm was firmly adherent to the surrounding vital structures. Although it was obvious that operation would not have been feasible in the presence of the complications that had developed, the treatment of these anomalies by surgery earlier in the course of this patient's life might have been successful.

DISCUSSION

This discussion will be limited to the subject of coarctation of the aorta, its diagnosis, complications, treatment, and a brief review of the literature.

Bonnet[6] classified coarctation of the aorta into the infantile and adult types, corresponding to their occurrence in the two age groups. In the infantile type, there is a diffuse narrowing of the isthmus of the aorta between the subclavian artery and the point of entrance of the ductus arteriosus. This type is usually associated with patent ductus arteriosus and other congenital malformations within the heart, which are usually not compatible with life, death commonly occurring within the first few weeks.

In the adult type the constriction is located at the point of insertion of the ductus arteriosus into the aorta, and is presumably due to the same process that causes normal obliteration of the ductus.[14] For this reason, this localized constriction of the aorta is usually associated with closure of the ductus and the development of extensive collateral arterial circulation. The immediate prognosis in this type is favorable, depending upon the volume of collateral blood supply in relation to the degree of constriction in the aorta.

On the basis of the anatomic and the functional differences between the two types one would not expect to see very frequently a patent ductus arteriosus with coarctation of the adult type, as was found in the case presented. A review of the literature bears out this point. Abbott's series[2] of 1000 cases of congenital heart disease contained 262 cases of patent ductus arteriosus and 178 cases of coarctation of the aorta. Of the latter, 70 were analyzed and only 6 cases were found to have a patent ductus. Evans[9] reported 28 cases, 17 of which had a patent ductus. Of these, only 2 patients (aged 8 years and 30 years) were in the age group past early infancy. Isolated cases have also been reported by Ulrich,[16] Graham and Kerr,[12] Fagin and associates,[10] and a few others.

The diagnosis of coarctation of the aorta should present no difficulty. The patient will usually have a high blood pressure in the arms with a lower pressure in the legs, and a systolic murmur of the heart transmitted to the back. X-ray examination of the chest may show notching of the ribs. Contrast roentgen

visualization of the great vessels will often demonstrate the point of constriction in the aorta.[11]

The prognosis of this disease is grave. In Abbott's series[1] of subjects above the age of 2 years, the average age at death was 32 years. Over 40 per cent die between the ages of 16 years and 30 years. There are three common complications which are usually responsible for the fatal outcome in these patients: aneurysm of the aorta with rupture, infection of the wall of the vessel with bacteremia, and hypertension with intracranial hemorrhage.

The development of an aneurysm might be expected to occur proximal to the coarctation, the segment of aorta which receives the greatest impact with each cardiac systoli. However, a more common site for this complication is at, or distal to, the point of stricture. Abbott[3] reported 14 cases of aneurysm, 13 of which were located at, or just below, the point of constriction. The pathologic changes in the wall of the aorta, with kinking and wrinkling producing eddy currents, are reasons which have been offered to explain this occurrence. The aneurysm may eventually rupture into the pericardium, the esophagus, the pleural cavity, or the bronchial tree.

Infection accounts for over 7 per cent of the deaths. It commonly results in damage to, and weakening of, the wall of the aorta, with the formation of a mycotic aneurysm. Although healing has been reported to occur, rupture of the aneurysm is a frequent fatal consequence. Before the introduction of chemotherapeutic agents into the medical armamentarium, the end result of infection of the blood stream in patients with congenital anomalies of the heart and great vessels was uniformly fatal. Today chemotherapy offers some hope to these patients if treatment can be instituted before the tissue damage has caused secondary complication. However, there are many problems which often present themselves for solution. Chief among these are drug toxicity, resistance of the infecting organism to the chemotherapeutic agent, recurrence of infection, and, finally, complications resulting from discharge of infected emboli. The recent discovery of coronamide, which acts to maintain high blood levels of penicillin by increasing its retention after administration, has eliminated to a great extent the problem of resistance of organisms ordinarily sensitive to penicillin.

Finally, hypertension of the arteries of the head, neck, and upper extremities often causes the patient disturbing and frequently incapacitating symptoms, such as headache and attacks of fainting. The secondary atheromatous and degenerative changes which later develop in the cerebral vessels may result in brain hemorrhage early in adult life. Early cardiac failure may also occur in these patients.

It is interesting that although the disease was described by Morgagni as early as the year 1760, it was not until 1945 that treatment was forthcoming. Crafoord[8] and Gross,[13] each independently, described a direct approach aimed at the surgical correction of the deformity. They resected the coarctated segment of the aorta and performed an end-to end suture anastomosis. Their reported results are very gratifying, offering new hope for a cure for a previously irremediable disease. The operation is best suited to coarctation of the adult type, and should be done at an early age. The procedure becomes progressively more difficult in patients past teen age. Where resection is not feasible, as in

the older age group, or in those patients whose coarctation extends over a long segment of the vessel (infantile type), a shunting procedure can be done by anastomosing the subclavian artery, or the carotid artery, into the aorta distal to the point of constriction.[7] This may relieve to a great degree the excess work of the heart, and abolish the symptoms referable to cerebral hypertension.

More recently experimental work[4] has indicated that free autogenous grafts of vein or artery may be used to replace resected segments of diseased aorta. This new concept may soon be extended to include homologous vein and artery grafts in the treatment of this and other diseases of blood vessels where resection is indicated, yet continuity of the vascular trunk must be preserved.

Since the successful treatment of this condition has been reported, interest in this and other congenital anomalies of the great vessels has increased. Ligation, or more recently section and suture, is now the generally accepted treatment for patent ductus arteriosus. Surgical interruption of congenital vascular rings around the trachea has also become the accepted therapy for these anomalies, now that the recognition of this condition has become more common. Likewise, by the early recognition of the syndrome of coarctation of the aorta, and with the knowledge of the ultimate grave prognosis of this disease, surgical treatment may be extended to these patients before the development of frequently fatal complications. It is hoped that this report will serve the purpose of stimulating further interest in the treatment of this condition, as well as to emphasize the seriousness of the disease, as demonstrated by the case presented.

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Surgical Technique

THE SURGICAL CORRECTION OF CONGENITAL EVENTRATION OF THE DIAPHRAGM IN INFANCY

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EVENTRATION of the diaphragm is characterized by the presence of an intact leaf of the diaphragm, which, however, has an abnormally high position. In the congenital variety of eventration there is no apparent cause for the abnormality. The condition is rare and is usually symptomless. However, in the occasional case, particularly in infants, there may be definite symptoms associated with eventration of the diaphragm and it then presents an interesting problem in therapy. A case of congenital eventration of the diaphragm in a 6-week-old male infant with successful outcome is herein reported.

REVIEW OF THE LITERATURE

Petit[1] was the first to describe the condition. A case report with post-mortem findings appeared in his writings published in 1774. Isolated case reports appear subsequent to this publication, but in 1913 Bergmann[2] reviewed the literature up to that date and collected 31 cases. In 1916 Bayne-Jones[3] reported on 45 cases, including one of his own. Korns,[4] in 1921, was able to collect 55 cases from the world's literature and in 1935 Reed and Borden[5] reported on 183 cases, most of which were in adults. During the recent war Kinzer and Cook[6] found congenital eventration of the diaphragm in 35 instances in 412,149 inductees who received routine roentgenograms of the chest. The eventrations were mostly on the left side and were entirely symptomless.

The number of cases reported of eventration in infants is extremely small. Up to 1916 Bayne-Jones[3] found 3 cases; in each instance the diagnosis was made at post-mortem examination. Reed and Borden[5] found only 5 cases of eventration in infancy and from 1935 to 1948 9 additional cases have been reported. This probably does not represent the true incidence of the disease because many cases are not recognized and, in addition, there are probably a number of cases that have been observed but not reported.

The preponderance of involvement of the diaphragm has been on the left side in practically all reports. In 183 patients of all ages, Reed and Borden[5] found only 18 eventrations on the right side.

As far as can be determined in a survey of the literature, operative repairs of the diaphragm have been done in only seven cases.[8] In all of them the diaphragm has been lowered by means of excision of a segment of the diaphragm or by plication. Most of the cases in the past have been approached through the thorax. In the cases reported, one patient died three days after operation and in

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the others, two obtained partial and three complete relief. The ages of these patients varied from 10 to 44 years. Bisgard[7] reported recently a case of eventration of the diaphragm in a male infant, 6 weeks of age, which was handled by means of a transthoracic approach with plication of the diaphragm. As far as can be determined this was the first reported case of surgical cure of this condition in an infant. The case reported here then constitutes the second such successful case.

CASE REPORT

R. M. (No. 779486), a 6-week-old, white male infant, was admitted to the University Hospital on June 9, 1947, because of spells of dyspnea and cyanosis on feeding and crying. The baby's mother first noted the breathing difficulty on the second postnatal day. Because of these bouts of cyanosis and dyspnea the baby had failed to gain weight at a proper rate. The prenatal period was essentially normal but the mother was in labor fifteen hours and delivery of the baby was by means of forceps. The baby was cyanotic for a considerable length of time immediately after birth and oxygen was needed to combat the cyanosis.

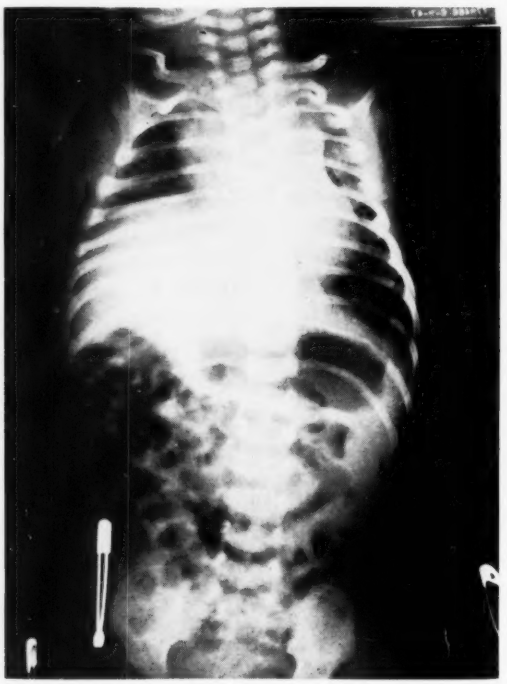

Fig. 1.—Preoperative roentgenogram of chest showing high position of right diaphragm and mediastinal shift.

Examination on admission revealed the baby to be well developed, somewhat undernourished, and in no acute distress. The respirations, however, were rapid and when the baby cried or took feedings he developed cyanosis. The entire right thorax anteriorly had a hyperresonent note to percussion, while over the left apex the note appeared to be somewhat dull. Beginning at the anterior axillary line, the right chest was dull to percussion, both laterally and posteriorly, and on auscultation the breath sounds were decreased on the right side posteriorly. In addition, there were occasional râles on expiration and inspiration on the right side posteriorly. The Wassermann tests of the blood of both baby and mother were negative.

The chest roentgenogram (Fig. 1) showed the position of the liver to be high in the right thorax but there was no evidence of bowel in the chest cavity. There was considerable

shifting of the mediastinum to the left. Fluoroscopic examination of the chest revealed definite paradoxical movements of the liver on the right side during respiration. The diagnosis was either eventration of the right diaphragm or possibly a diaphragmatic hernia. These examinations failed, however, to differentiate definitely between the two. Because of the high position of the right diaphragm with atelectasis of the lung and the cyanosis on feeding and crying, it was felt that exploration should be done and the defect in the diaphragm corrected. Operation was undertaken on June 18, 1947.

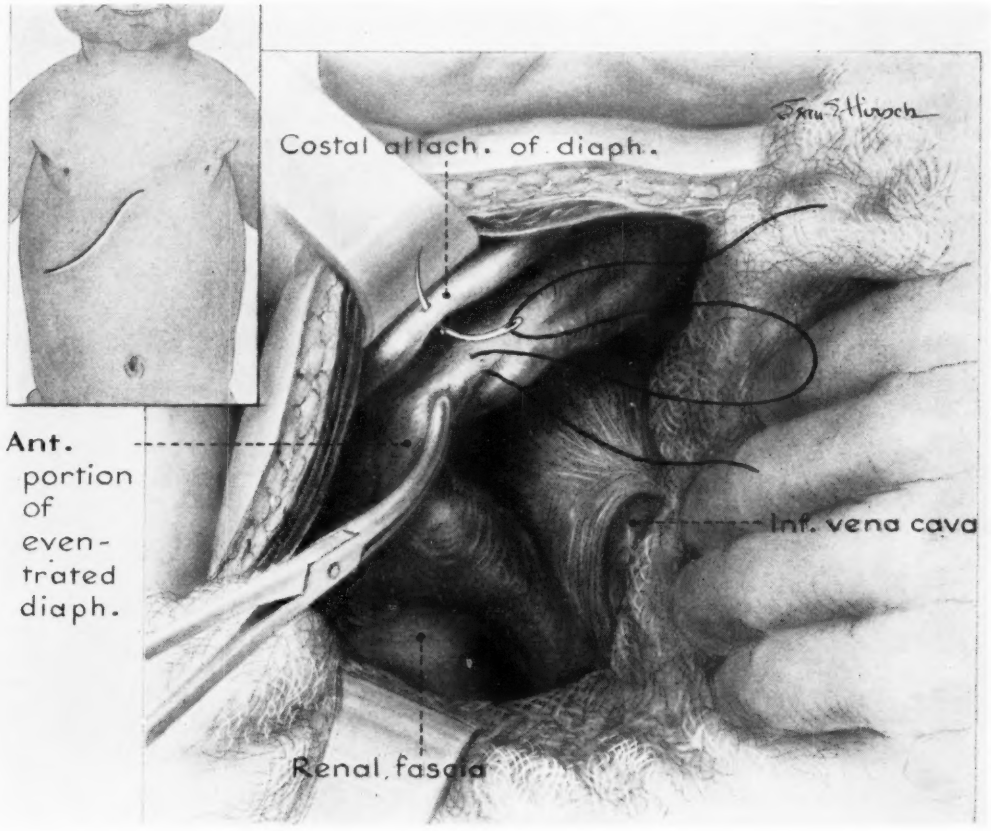

Fig. 2.—Anterior portion of eventrated diaphragm is sewn to costal attachment of diaphragm. *Insert*, Subcostal incision utilized in the case reported.

Operative Procedure.—The anesthesia utilized was ½ per cent procaine with two drops of $\frac{1}{1000}$ adrenalin hydrochloride to the ounce for local infiltration of the anterior abdominal wall. This was supplemented with oxygen and cyclopropane administered through a tight-fitting mask. The abdomen was opened through a subcostal incision in the right upper quadrant (Fig. 2). On entering the peritoneal cavity it was noted that the diaphragm was high in position with the apex reaching to the level of the second interspace anteriorly. As there were, however, no defects in the diaphragm, the diagnosis of eventration of the right diaphragm was established. The left diaphragm was low and bulged convexly inferiorly indicating the presence of considerable emphysema of the left lung. The right diaphragm, although high in position, seemed to be normally developed with considerable muscular tissue at the circumference of the diaphragm and a rather thick central tendinous trefoil. It was felt that the child's symptoms of cyanosis and dyspnea were due mainly to atelectasis of the right lung and mediastinal shift and an attempt should be made to correct the high position of

the diaphragm. Accordingly, the anterior portion of the diaphragm was grasped with forceps and brought down and attached to the circumference of the costal margin of the diaphragm anteriorly (Fig. 3). This first step produced considerable lowering of the diaphragm but the posterior portion still was high. This posterior portion of the diaphragm was then grasped with the forceps brought down and sewn to the crus of the diaphragm medially and to the renal fascia laterally (Figs. 3 and 4). The abdominal surface of the right diaphragm was now at the level of the costal margin anteriorly and the transverse process of the twelfth rib posteriorly. The suture material utilized was 000 Deknatel silk.

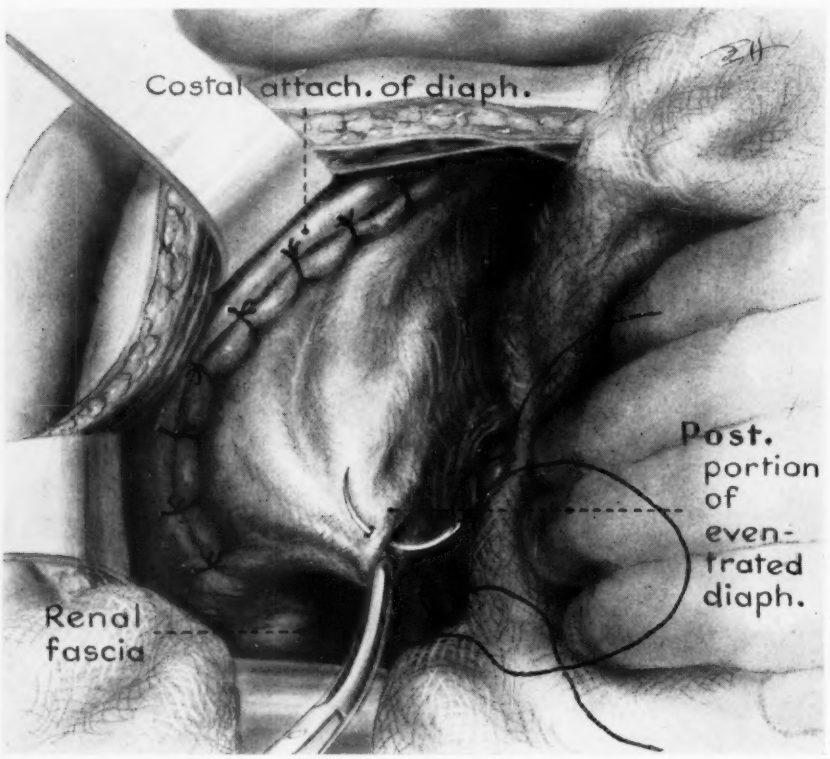

Fig. 3.—Posterior portion of eventrated diaphragm is sewn to crus of diaphragm.

The liver, as in the cases of diaphragmatic hernia, had no peritoneal attachment to the diaphragm, consequently, it could be easily mobilized and brought down inferiorly and on to the anterior abdominal wall so that the repair could be carried out on the diaphragm without interference from the liver. When this stage of the operation was completed it was felt that it would be best not to suture all the layers of the anterior abdominal wall at this time because undue pressure would be exerted on the newly constructed diaphragm. The skin was thus brought over the abdominal viscera without any attempt being made to close the muscular and fascial layers of the abdominal wall (Fig. 4). The patient withstood this operation nicely. A nasal tube was left into the stomach for decompression purposes for twenty-four hours. The position of the diaphragm as indicated by the roentgenograms immediately postoperatively showed there was considerable lowering of the right diaphragm. There was also considerable clearing of the atelectatic changes in the lung and improved position of the mediastinum (Fig. 5).

The patient was discharged from the hospital on July 7, 1947, with the wound well healed but with considerable bulging of the incision as was to be expected from the fact that only skin was utilized for closure. On June 20, 1948, approximately one year later, after

considerable growth had occurred, the infant was readmitted to the University Hospital and a repair of the ventral hernia carried out. The abdominal layers on either side of the incision came together nicely and without undue tension. X-ray pictures of the diaphragm subsequent to this repair of the ventral hernia showed that the right diaphragm remained in good position as can be seen from Fig. 6.

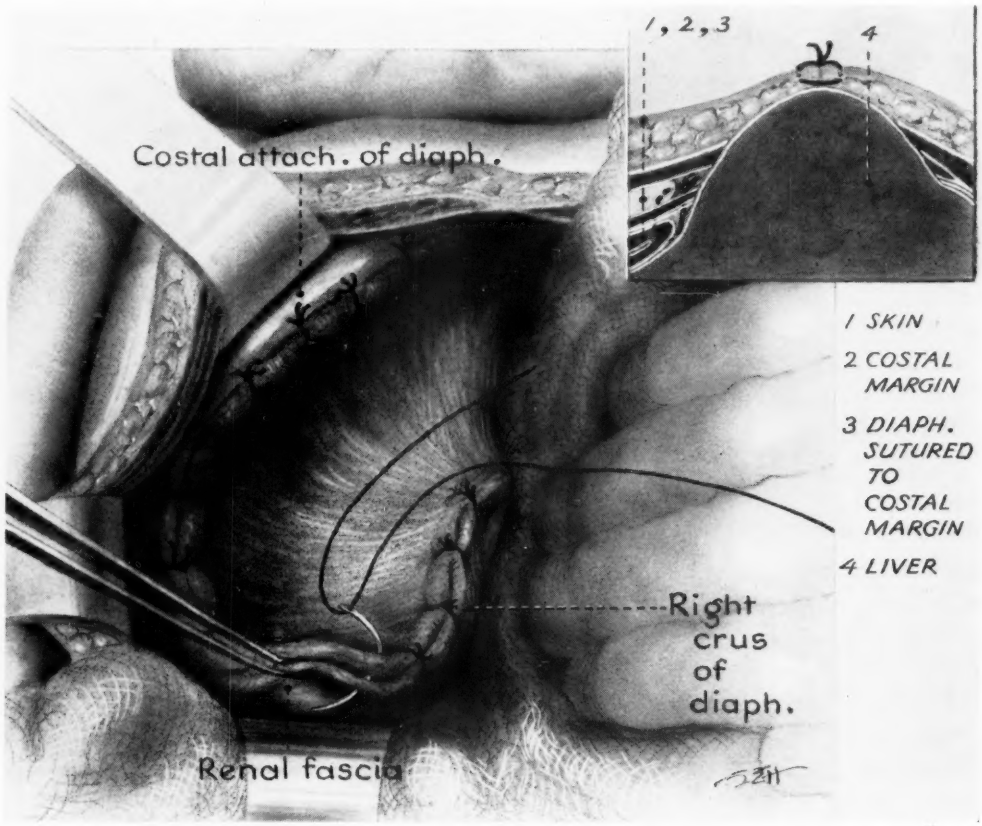

Fig. 4.—Suturing of posterior portion of eventrated diaphragm to renal fascia completed. Note thickness of diaphragm and renal fascia. *Insert,* Abdominal wall closure; skin and subcutaneous tissue.

DISCUSSION

There has been no satisfactory explanation of the cause of congenital eventration of the diaphragm. Reed and Borden[5] reviewed the pathologic descriptions of the cases reported and have found that only in a few instances have there been adequate descriptions of the tissues. In these cases the diaphragm was a thin, translucent membrane and had only a few atrophied muscle fibers at the periphery of the diaphragm. Sailer and Rhein[9] found numerous muscle fibers at the periphery of the diaphragm in the cases they studied, but in most instances the muscle fibers were markedly atrophied. Motzfeldt[10] reported fatty infiltrations and atrophy with fatty changes in the muscle of the eventrated diaphragm. In most instances the phrenic nerve has been without unusual changes. However, Morrison[11] described a number of

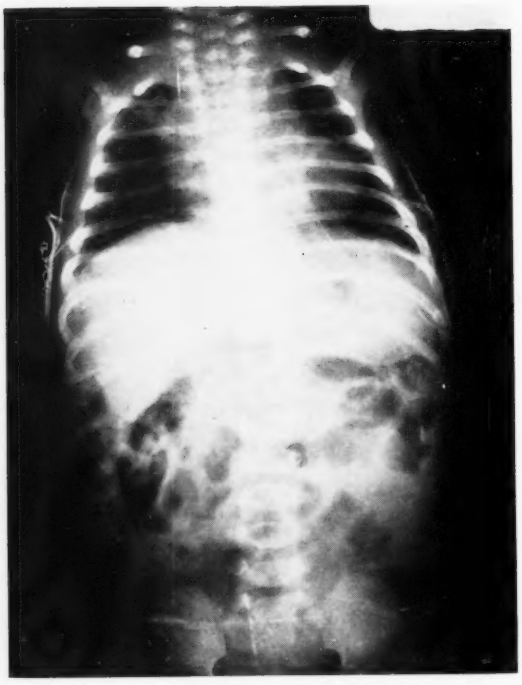

Fig. 5.—Postoperative (three days) roentgenogram of chest. Note lowering of right diaphragm and position of mediastinum.

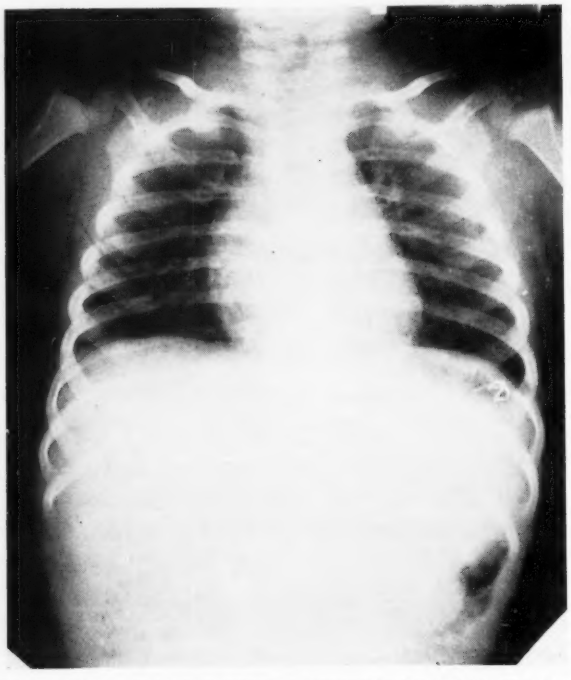

Fig. 6.—Roentgenogram of the chest one year postoperatively. Note position of right diaphragm and mediastinum.

cases in which there appeared to be fibrous tissue masses involving the phrenic nerve, which could thus explain the eventration.

Other congenital abnormalities are usually associated with the eventration of the diaphragm as in Korns'[4] case where there was lobulation of the lung and in Rosenfeld's[14] case where there was complete inversion of the stomach. Other abnormalities which have been reported are nonrotation of the colon, megacolon, hypospadias, and harelip.

Although the symptoms described in this case were essentially associated with the respiratory tract, Reed and Borden[5] also called attention to the fact that there may be symptoms associated with circulatory and gastrointestinal systems. Those symptoms which are associated with the gastrointestinal tract are, in order of frequency, abdominal pains, vomiting, dyspepsia, constipation, dysphagia, and heartburn. The cardiac symptoms that have been noted consist of palpation, tachycardia, and cyanosis.

The diagnosis of the condition is usually established by means of x-ray examination of the chest, although in some instances it may be difficult to differentiate between eventration of the diaphragm and a diaphragmatic hernia. Introduction of oxygen into the peritoneal cavity with subsequent chest plates or fluoroscopic examination of the chest in the upright position may help to differentiate between the two conditions. However, it must be remembered that there have been a number of fatalities with the use of oxygen[12] in cases of diaphragmatic hernia, so that this procedure must then be used with caution.

In the case described, the surgical approach has been different in a number of aspects from other cases previously reported. The diaphragm is lowered by "tenting" caudadward in several planes and it is then fixed in this new position by suturing the diaphragm to the costal margin anteriorly, and to the crus of the diaphragm and renal fascia posteriorly. It is surprising to find out how firm and thick the renal fascia is in infants of this age and how well this substance will permit suturing. The same observation has been made by Weinberg[13] in the repair of congenital hernias of the diaphragm. The plan of closure of the abdominal wall in two stages also permits stabilization of the diaphragm until readjustment of the viscera to the small peritoneal cavity has occurred and prevents undue pressure upon the newly sutured diaphragm until healing is complete.

It was surprising to note the thickness of the diaphragm, for in the adult cases of eventration, the diaphragm is tissue-paper thin and translucent. It would be difficult to do a surgical repair on such thinned and atrophied diaphragms. The well-developed diaphragm in infancy, on the other hand, lends itself nicely to surgical correction.

CONCLUSION

A case of eventration of the diaphragm in a 6-week-old male is reported. The malposition of the diaphragm was apparently responsible for the development of cyanosis and dyspnea while the infant was feeding or crying. The successful surgical correction of the eventration was accomplished through an

abdominal approach utilizing a method of tenting and fixation of the diaphragm, as well as a two-stage closure of the abdominal wall.

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Editorial

The Increasing Frequency of Injury of the Common Bile Duct and Hepatic Duct

A THOROUGH knowledge of the anatomy of the biliary tract, including the anomalies which may be found, accurate exposure, and lighting of the operative field are absolute necessities for safe biliary surgery. In the absence of these factors, serious injuries of the common bile duct, the hepatic artery and its main branches, and the portal vein may easily occur. Injuries occasionally will occur under the best of conditions, but they may be recognized and corrected immediately much more easily and with better results than they can be at a secondary operation.

The importance of previous operations on the biliary tract as a cause of stricture of the common bile duct is indicated by the fact that all but two of the 165 patients on whom one of us (W. W.) has operated for such stricture previously had undergone an operation of the biliary tract. The fact that profuse drainage of bile from the wound, and jaundice, dark urine, or acholic stools, or several of these findings occurred within a few days after the original operation supports the contention that the bile duct was injured at the original operation with resulting stricture. A review of the literature will disclose only a few cases in which extensive contracture of the bile ducts has occurred as a result of infection in the biliary tract or in adjacent structures and in which operation has not been performed previously. Stricture of the bile ducts is an extremely serious condition, but it is by no means hopeless even after several unsuccessful attempts at repair.

From 1924 to 1939 inclusive, one of us (W. W.) operated in 98 cases of benign stricture of the common bile duct at the Mayo Clinic. In 18 of the 98 cases the patients had a complete stricture of the extrahepatic ducts. In the remainder, biliary intestinal continuity was established at operation. Ten patients died in the hospital after the operation. In 4 of the cases, death was due to postoperative hemorrhage. In these 4 cases, the operation was performed before the advent of vitamin K. A follow-up study made in 1940 revealed that 52 of the 98 patients were living and well.

From January, 1940, to July, 1942, and from January, 1946, to January, 1948, operation was performed in 67 additional cases. In these cases, a total of 126 operations had been performed on the biliary tract before the patients came to the clinic. In 2 of the cases (3 per cent), the patients died postoperatively. In one of these cases, death was due to hepatic and renal insufficiency; in the other case, it was due to pelvic and subphrenic abscesses which followed disruption of a hepaticoduodenal anastomosis.

In 51 of the 67 cases, only one operation for the repair of a stricture of a bile duct was performed at the clinic. Follow-up data were obtained in 41 of

the 51 cases. The result of the operation was satisfactory in 31 of the cases. Two of the patients died of disease of the liver after they had been dismissed from the clinic but they did not require a subsequent operation on the bile ducts. In 14 of the 67 cases, at least one subsequent operation was performed on the biliary tract at the clinic. Follow-up data are available in 10 of the 14 cases. Six of the 10 patients have not had any symptoms of biliary obstruction since they left the clinic; 3 have had mild symptoms of biliary obstruction at infrequent intervals, and 1 patient is free of symptoms after the third operation at the clinic.

In general, it may be said that one operation for the repair of stricture of the bile duct has produced a satisfactory result in 60 per cent of cases. In an additional 10 per cent of cases, the patients will have an occasional attack of recurrent cholangitis but they will not require another operation for relief of the stricture. Twenty per cent of the patients will require another operation because of the recurrence of biliary obstruction. About 10 per cent of the patients will die of disease of the liver within a few years after the first operation is performed.

If possible, the ends of the duct should be anastomosed over a catheter which is permitted to extend into the duodenum and is anchored in place for three to six months with a silk suture which passes through a button on the skin, as advocated by McArthur. Intestinal peristalsis will pull out the catheter when the anchoring silk thread is removed. The results of this operation usually are excellent. If the stricture is extensive, it may be necessary to perform a choledochoduodenostomy or a hepaticoduodenostomy. In performing either of these operations, accurate anastomosis is essential. The anastomosis is usually but not always made over a Mayo-Sullivan tube in order to prevent the circular contracture that occurs in varying degree in the following three months. Lately, we have used a Mayo-Sullivan tube made of polythene, a polymer of ethylene, which is nonwettable by bile. This tube is maintained at the site of the anastomosis by a silk thread which is brought to the exterior of the body and anchored to a button.

An accurate anastomosis is the most important factor in preventing recurrent stricture and ascending cholangitis. Infection does not occur unless obstruction is present. In cases in which chills, fever, and pain occur with or without jaundice after an operation has been performed for stricture of the common bile duct or hepatic ducts, an exploratory operation discloses a stricture of small caliber or a contracture at the site of the anastomosis. Tubes are useful as splints at the site of the anastomosis and to prevent contraction of the scar tissue during the first several months after the operation. After that they may be a hindrance to the passage of bile and may become obstructed with bile salts and bile pigment. This has happened frequently in tubes made from Vitallium or rubber, but so far it has not been evident in tubes made of polythene.

Penicillin administered with or without streptomycin is preferable to the sulfonamide drugs in the treatment of cholangitis, but it will produce only temporary relief until the obstruction is removed. Sulfadiazine administered for a

few days or a week each month will sometimes control residual cholangitis. Vitamin K has been of utmost importance in reducing the danger of postoperative hemorrhage.

Strictures of the bile ducts, which seem to be increasing each decade, and especially since the war, are nearly always the result of injury of the bile ducts at the time of operation. Careful exposure and dissection and an accurate knowledge of the regional anatomy are necessary to avoid these injuries. Although the dangers of biliary operations for stricture are less and the results better than they were formerly, stricture of the bile ducts remains a very serious condition but is never a hopeless one.

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Recent Advances in Surgery

Conducted by Alfred Blalock, M.D.

FACTITIAL (IRRADIATION) PROCTITIS

A Clinicopathologic Study of 200 Cases

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THE introduction of radium and roentgen rays as therapeutic agents is responsible for a previously nonexistent type of rectal and sigmoidal lesion. Irradiation may affect normal tissues as well as those which are diseased. Experience has proved that benign lesions of the rectum, sigmoid, and small bowel may develop in some patients after irradiation of extrarectal pelvic conditions, usually carcinoma of the uterine cervix, and that these lesions may produce symptoms similar to those caused by carcinoma.

PRIOR STUDIES

The term "factitial proctitis" was used for the first time by Malmgren and one of us[1] in 1930 to designate the benign lesions which sometimes involve the walls of the rectum or sigmoid after the use of radium or roentgen rays in the treatment of lesions of pelvic structures adjacent to the rectum. "Radiation proctitis" and "radium burn of the rectum" also have been employed by other authors to designate this condition.

The first report of a rectal lesion which developed after irradiation of an extrarectal process was made by Füth and Ebeler[2] in 1915. The report of Malmgren and one of us[1] in 1930 concerning 65 patients who had factitial proctitis contained the first published description of the proctoscopic appearance of the lesions. In 1937 Bacon[3] reported thirty-nine cases. He divided the pathologic changes in the rectum into three stages: (1) the stage of congestion or hyperemia, (2) the stage of ulceration, and (3) the stage of formation of stricture. In 1942 Aldridge[4] reported thirty-eight cases. He described three types of the condition: (1) acute localized proctitis or proctosigmoiditis, (2) ulceration of the mucosa and wall of the intestine, and (3) perirectal fibrous tissue. These and other reports indicate that factitial proctitis now is considered to be a clinical entity.

NATURE OF PRESENT STUDY

The present study includes the records of 200 patients who received irradiation therapy for benign or malignant extrarectal conditions, and in whom a

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factitial lesion was discovered at proctoscopic examination. The majority of these patients came to the Mayo Clinic because of symptoms referable to the rectum, but in some the rectal condition was of minor importance. Some patients had received irradiation elsewhere and were examined at the clinic only in order to determine if additional irradiation should be administered.

In this study, four gross specimens were studied, as were also twenty-four specimens obtained at biopsy from patients other than those from whom the four gross specimens were removed. The tissue had been preserved in formalin for periods ranging from ten months to fourteen years. Blocks of tissue which included the entire thickness of the bowel and a portion of the perirectal tissue were examined microscopically. Fixed sections, prepared by the frozen section technique and stained with hematoxylin and eosin, were made. An amyloid stain was used on several sections from one lesion.

All patients were women; 186 (93 per cent) had received irradiation treatment of a malignant lesion situated in an extrarectal pelvic organ. The remaining 14 (7 per cent) patients received irradiation of a benign pelvic process. The conditions for which irradiation was administered are shown in Table I.

TABLE I. FACTITIAL (IRRADIATION) PROCTITIS: LESIONS FOR WHICH IRRADIATION WAS ADMINISTERED IN 200 CASES

LESION OR CONDITION	CASES	PER CENT
Carcinoma of cervix uteri	165	82.5
Carcinoma of fundus uteri	12	6.0
Carcinoma of ovary	6	3.0
Carcinoma of vagina	3	1.5
Benign menometrorrhagia	6	3.0
Benign tumor of uterus	5	2.5
"Ulcer" of cervix	2	1.0
Persistent backache	1	0.5
Totals	200	100.0

Factitial rectal and sigmoidal lesions occurred after irradiation with radium alone, roentgen rays alone or after a combination of both radium and roentgen rays. Eighty-two (41 per cent) patients received all irradiation at the Mayo Clinic, 14 (7 per cent) received a portion of it at the Mayo Clinic, and 104 (52 per cent) received all irradiation elsewhere. The 96 patients who received either all or a portion of the irradiation at the Mayo Clinic had malignant extrarectal lesions.

SYMPTOMS

Because of the nature of the primary pelvic disturbance, patients such as these under discussion become very observant. They watch their daily habits carefully. When a patient who has had pelvic irradiation therapy complains of symptoms concerning the bowel, the following questions should arise in the mind of the physician.

Are the signs and symptoms caused by an extension of the extrarectal process, with secondary involvement of the bowel? Are they caused by a separate primary rectal or sigmoidal malignancy? Are they caused by the development of a factitial lesion in the bowel?

Although the great majority of factitial lesions occur in women, they have developed in men after the irradiation of tumors of the bladder and prostate gland.

In order of frequency of occurrence, symptoms were rectal bleeding, diarrhea, rectal pain, rectal tenesmus, constipation, and abdominal cramping. In the majority of cases various combinations of these symptoms were present or had been present at some time.

Rectal bleeding was a predominant symptom in 153 (76.5 per cent) of the 200 cases. In most of the remaining 47 patients who had not noticed rectal bleeding, slight bleeding from the injured mucosa was observed on examination. In 90 per cent of the patients with bleeding, the blood was slight to moderate in amount, and in the remainder it was marked. Most patients who mentioned rectal bleeding had no ulceration in the rectum or sigmoid. The bleeding in these instances arose from small telangiectatic vessels in the mucosa.

Diarrhea was a symptom in 71 (35.5 per cent) of the 200 patients. We distinguished between true diarrhea and a rectal discharge usually accompanied by rectal tenesmus and which, because of its frequency of occurrence, simulated diarrhea. Brief episodes of diarrhea which followed irradiation were not included. In about 90 per cent of the cases, diarrhea was mild to moderate in degree; in the remainder it was marked. It was most common when the bowel was ulcerated or contracted.

Rectal pain occurred in 64 (32 per cent) of the 200 patients. Usually, it was predominant in patients who had rectal or sigmoidal ulceration. The site of pain varied; patients had difficulty in describing it accurately. Because the nerve supply cephalad to the pectinate line is autonomic, it is difficult for patients to localize discomfort accurately in that area. One-half of the patients with severe rectal pain had pelvic or perirectal infiltration.

Four patients had a rectovaginal fistula. When first examined, 3 of them had experienced constant severe rectal pain prior to the development of the fistula. The pain was relieved after the fistula appeared.

Rectal tenesmus was a symptom in 49 (24.5 per cent) patients. These patients complained of a constant and urgent desire to defecate, and only bloody mucus was expelled as a rule. An uncomfortable sensation persisted after expulsion of the discharge, as if the evacuation had been incomplete. Tenesmus was complained of frequently by patients who had rectal or sigmoidal ulceration, but 3 of the 5 patients who complained chiefly of rectal tenesmus had proctitis or sigmoiditis without ulceration or stricture. In such instances, the inflammatory process usually was severe.

Constipation was complained of by 38 patients (19 per cent), and abdominal cramps by 31 (15.5 per cent). The incidence of abdominal cramps was highest in the patients who had rectal or sigmoidal stricture.

Eleven patients complained of urinary distress. In each of these patients factitial cystitis, with or without ulceration, was found on cystoscopic examination.

Symptoms referable to the rectum occurred during the course of irradiation in 8 (4 per cent) of the 200 patients; they appeared in 55 (27.5 per cent) during

the first week after termination of irradiation; they developed in 23 (11.7 per cent) during the second through the fourth week; by the end of the third month symptoms had appeared in slightly more than one-half (52.5 per cent) of the 200 patients. In 35 (17.8 per cent) of the 200 patients symptoms developed during the next three months; in 21 (10.7 per cent) patients symptoms developed during the third three-month period; and in 10 (5.1 per cent) patients the symptoms did not appear until the fourth three-month period. Among the remaining patients symptoms developed in 13 (6.6 per cent) during the second year; in 8 (4 per cent) during the third; in 4 (2 per cent) during the fourth; in 1 (0.5 per cent) during the fifth; and in 1 patient symptoms did not appear until 6¾ years after completion of irradiation. Four of the 200 patients did not mention symptoms referable to the bowel. These 4 were examined proctoscopically because the clinician had noted abnormal signs on digital examination of the rectum during a routine examination.

Of the 27 patients in whom symptoms developed one year after irradiation, 20 had proctitis or sigmoiditis without ulceration or stricture, 4 had ulceration with stricture, and 3 had stricture with ulceration. The time of onset of symptoms appears to have no relationship to the type of factitial lesion.

SITE OF LESION

The middle third of the anterior wall of the rectum was the most frequent site of factitial lesions. Of the 66 instances of ulceration, 37 (56 per cent) involved that area. In two cases a lesion was confined entirely to the right rectal wall, in one only the left rectal wall was involved, and in another there was a lesion on the anterior wall and a separate lesion on the posterior wall. Some lesions involved the entire circumference of the bowel. Strictures were found in each segment of the rectum and sigmoid except the lowest third of the rectum.

The location of the factitial lesion in the 200 cases was as follows: in 13 patients it was confined entirely to the lowest third of the rectum; in 75, to the middle third; in 37 to the upper third; in 27 to the rectosigmoidal area; in 18 the lesion was found only in the sigmoid. The entire rectum was involved diffusely in 10 instances. In 7 the involvement included the middle and upper third parts of the rectum, and in 2 the upper third part of the rectum, the rectosigmoid, and the sigmoid were involved. In the remaining 3 cases the midrectum, rectosigmoid, and sigmoid were concomitantly involved by factitial proctitis and sigmoiditis, and the upper third part of the rectum was normal. Thus, the lowest third portion of the rectum was involved in 26 (13 per cent) patients, the middle third portion in 103 (51.5 per cent), the upper third portion in 56 (28 per cent), the rectosigmoid in 36 (18 per cent), and the sigmoid in 22 (11 per cent).

TYPES OF LESIONS

According to morphologic characteristics and pathologic anatomy, six types of factitial lesions are observed. Examples of all types of factitial lesions except those complicated by fistulas were found in the sigmoid, rectosigmoid, as well as in the rectum. Thirteen of the patients in whom the lesion was located at or

above the level of the rectosigmoid had received only intravaginal or intrauterine irradiation with radium.

Proctitis or Sigmoiditis Without Ulceration or Stricture (Table II).—Acute lesions in these conditions are characterized by edema, hyperemia, and excess secretion of mucus. Spasm of the wall of the bowel usually is associated. Edema and redness generally are most prominent during this stage. During the healing stage the mucosa is pale, and telangiectasis is seen. After complete healing patients may complain of rectal bleeding. The source of the blood is the delicate telangiectatic vessels. As a rule, the light swabbing of such an area with a cotton applicator is sufficient to cause oozing of blood.

TABLE II. FACTITIAL (IRRADIATION) PROCTITIS: NATURE OF LESIONS IN 206 CASES

LESION	CASES	PER CENT
Factitial proctitis or sigmoiditis without ulceration or stricture	106	53.0
Factitial proctitis or sigmoiditis with ulceration and without stricture	47	23.5
Factitial proctitis with rectovaginal fistula and without stricture	3	1.5
Factitial proctitis or sigmoiditis with stricture and without ulceration	24	12.0
Factitial proctitis or sigmoiditis with ulceration and stricture	19	9.5
Factitial proctitis with rectovaginal fistula with stricture	1	0.5
Totals	200	100.0

Proctitis and Sigmoiditis With Ulceration and Without Stricture (Table II).—In these conditions the factitial ulcer presents a characteristic appearance. Proctitis or sigmoiditis with telangiectasis is observed adjacent to the ulcer. In most instances the ulcer is oval or irregularly circular, and involves from one to all layers of the wall of the bowel. The base of the ulcer lies below the level of the margins and is covered with a tenacious, silvery gray membrane. In some instances, it is covered with dirty gray debris which can be removed. In several instances in this series we found that the base of an ulcer was a part of the vaginal wall. Ulcers may be observed at all periods of the disease. In some instances the ulcer bleeds profusely.

In our experience, the earliest diagnosis of a factitial ulcer was made two weeks after completion of irradiation. More than one ulcer was observed in only 4 cases. The single ulcer is one of the usual characteristics of the condition. The diameter of the ulcers varied from 0.5 to about 6 cm.

Proctitis or Sigmoiditis With Stricture and Without Ulceration (Table II).—In such a condition telangiectasis was always present in the mucosa. The strictures were characteristically smooth and symmetrically narrowed, except in the cases in which perirectal infiltration was present. In one-half of the cases the fibrous process was limited to only the wall of the bowel, as evidenced by its mobility. In the other half, the stricture was part of a dense pelvic mass which tended to immobilize the terminal portion of the bowel.

Twenty-four patients had this type of lesion. In 4 patients the area of greatest constriction was in the midrectum; in 6 it was in the upper part of the rectum; in 7 it was in the rectosigmoidal area; in 7 it was in the sigmoid. The

earliest diagnosis was made four months after completion of irradiation. The diameter of the strictured areas averaged 1 to 1.5 cm.

Proctitis or Sigmoiditis With Ulceration and Stricture (Table II).—Sometimes the ulcer involves only a small area. In other instances, a larger portion is involved; and in still others the entire stricture is ulcerated. Nineteen patients had this type of lesion. In 7 the area of greatest involvement was in the mid-rectum; in 3 it was in the upper part of the rectum; in 6 it was in the recto-sigmoidal area; in 3 it was in the sigmoid. The earliest diagnosis in this group was made two months after completion of irradiation. The average diameter of the lumen of the stricture was 1.5 cm.

Proctitis With Rectovaginal Fistula (Table II).—The fistulas were located in an area of factitial proctitis which extended various distances from the margins of the fistulas. They were lined by a grayish membrane. The smallest was 0.5 cm. in diameter and the largest was 2.5 cm. Contraction of the rectal lumen was seen in 1 patient.

Other Associated Conditions.—Various degrees of induration and fixation of the involved areas were noted in most of the cases. Perirectal infiltration was present in 110 (55 per cent) of the 200 patients at the time of the initial proctoscopic examination. Slight infiltration was present in 24 cases, moderate infiltration in 40, and marked infiltration in 46. The degree of perirectal infiltration did not appear to have any relationship to the severity of symptoms. The incidence of symptoms of obstruction was no greater among the patients with stricture and extensive perirectal infiltration than it was among those with stricture in a freely movable bowel. Frequently the examiner finds that the "feel" of perirectal infiltration is similar to that imparted by a malignant process. Often the problem of determining the presence or absence of cancer is difficult, but it must be decided in every patient as the physician contemplates the type of treatment which is required.

ROENTGENOLOGIC EXAMINATION

Roentgenologic examination of the colon by administration of a barium enema was performed for 37 of the 200 patients. In 10 patients a constriction with or without mucosal ulceration was observed in the sigmoid beyond the reach of the sigmoidoscope. In 3 patients the constriction observed during roentgenologic examination was identical to that visualized on proctosigmoidoscopic examination, and in each instance the roentgenologist was able to distinguish the lesion from carcinoma.

CLINICAL COURSE

One hundred thirty-nine of the 200 patients were examined only once with the proctosigmoidoscope. Sixty-one patients had follow-up examinations over periods of one month to fourteen years. Of these 61 patients, 43 had lesions which either healed during the period of observation, began to heal, or remained essentially the same as when first seen. In the remaining 18 patients there was an increase in the factitial changes.

In the cases of proctitis or sigmoiditis without ulceration or stricture, the edema and redness in the mucosa subsided after the acute stage. As healing progressed, the injured areas became paler and a slightly yellowish tinge developed. Also, telangiectasis developed and was the source of periodic rectal bleeding after healing of the lesion. In 7 of the patients with this type of factitial lesion there was an increase in the severity of the condition. Among these 7 patients the area of proctitis became more extensive in 1, ulceration without stricture appeared in 2, ulceration with stricture developed in 3, and in the seventh the rectal lesion remained the same as when first seen but areas of factitial ileitis appeared. This condition was observed during the performance of an abdominal operation.

The edema and hyperemia in the margin of the ulcers disappeared after the acute stage, and the surrounding collar of proctitis and sigmoiditis became less pronounced. As healing progressed, the size of the ulcers decreased. After healing was complete, the site of the ulcers was marked by paleness and scarring of the mucosa, in addition to vascular telangiectasis.

The ulcers in 2 of these patients became rectovaginal fistulas. The fistulas appeared three and one-half, and two years after diagnosis of the factitial disturbance. The ulcer in one of these patients was 4 by 6 cm. in surface dimensions when it was first seen. Gross involvement of the rectum by a pelvic malignant process was not found at examination either prior to or after the development of the fistulas. In another case a stricture developed when the ulcer healed.

While the patients with proctitis or sigmoiditis with stricture and without ulceration were under observation, rectal or sigmoidal ulceration developed in 4 and a rectovaginal fistula developed in 2. In 1 of these 4 patients who developed ulceration, the lesion healed while the patient was under observation. The ulcer and stricture in another patient were resected surgically; and the other two patients did not return for additional examinations. One of the rectovaginal fistulas was repaired surgically and the rectal stricture was resected. In another patient a marked rectal stricture became dilated without treatment during the ensuing eleven years.

Eight patients who had proctitis or sigmoiditis with ulceration and stricture had follow-up examinations. In 5 patients the ulceration healed; in the other 3 rectovaginal fistulas developed. These fistulas appeared three weeks, three months, and six months after diagnosis of the factitial ulcer.

Only 1 of the patients who had proctitis and rectovaginal fistula was examined more than once. In this instance, the examination was done ten months after diagnosis of the factitial lesion, and at the time of examination the fistula appeared to be closing spontaneously. Colostomy had been performed elsewhere prior to the time of the second examination.

CASES IN WHICH FACTITIAL PROCTITIS HAD BEEN MISTAKEN FOR MALIGNANT INVOLVEMENT OF THE RECTUM

Nineteen (9.5 per cent) of the 200 patients were referred to the clinic because their home physicians had made a clinical diagnosis of rectal carcinoma. Fourteen of these 19 patients had been told that the original pelvic malignant

process involved the bowel. The remaining 5 patients came to the clinic with a diagnosis of primary cancer of the rectum.

No evidence of either a primary or secondary malignant lesion of the bowel was found in any case. A typical factitial lesion of the rectum or sigmoid was present in each instance. Factitial proctitis or sigmoiditis without ulceration or stricture was present in 4 cases; factitial ulceration without stricture was present in 10; factitial proctitis or sigmoiditis with stricture and without ulceration was present in 1; and factitial ulceration with stricture was present in the remaining 4. The interval of time between completion of irradiation and the appearance of the rectal symptoms in these 19 cases varied from two weeks to six months.

PATHOLOGIC DATA

Gross Aspects.—As stated previously, 4 gross surgical specimens and 24 additional specimens taken for biopsy were studied. There was ulceration in 3 of the gross specimens, and stricture without ulceration in the fourth.

Patients who had gross ulceration had undergone irradiation of lesions of the uterine cervix. In 2 cases the lesion was cancer and in the other, an "ulcer." The onset of rectal symptoms was two months, four months, and eight months, respectively, after completion of irradiation. Resection of the injured portion of bowel was performed in 2 cases because of stricture and in the third case because of severe, repeated rectal hemorrhages. The interval of time between the completion of irradiation and resection was nine, nine, and eleven months, respectively.

The patient who had stricture without ulceration had undergone irradiation of a cancer of the uterine cervix. The onset of rectal symptoms occurred one month after the completion of irradiation. Colostomy had been performed soon after the onset of the symptoms arising from the rectum, but the stricture was not resected until thirteen years later.

Pathologic Anatomy.—

Specimen 1: This specimen consisted of 8 cm. of the rectosigmoid and sigmoid. In it was an area of ulceration 2 by 2.5 cm. in surface dimensions. The edges were well defined; the base was slightly ragged. The wall of the bowel was thickened and the lumen of the bowel was constricted in the region of the ulcer (Fig. 1).

Specimen 2: This specimen consisted of 15 cm. of the rectum, rectosigmoid, and sigmoid. Near each end of it was an ulcer 2.5 cm. in diameter. The ulcers were 10 cm. apart. The edges were well defined; the bases were slightly ragged. The wall of the bowel was thickened; there was narrowing of the lumen of the bowel in the region of each ulcer.

Specimen 3: This specimen consisted of 25 cm. of the anus, rectum, rectosigmoid, and sigmoid. Three centimeters cephalad to the anal margin was an ulcer 3 by 2 cm. in surface dimensions. Three centimeters internal to this ulcer was another area of ulceration 1.6 cm. in diameter. The edges of the ulcers were well defined. There was no narrowing of the lumen of the bowel.

Specimen 4: This specimen consisted of 8 cm. of the rectum, rectosigmoid, and sigmoid. Marked constriction of the lumen had occurred in the midportion of the diseased area, but there was no gross ulceration.

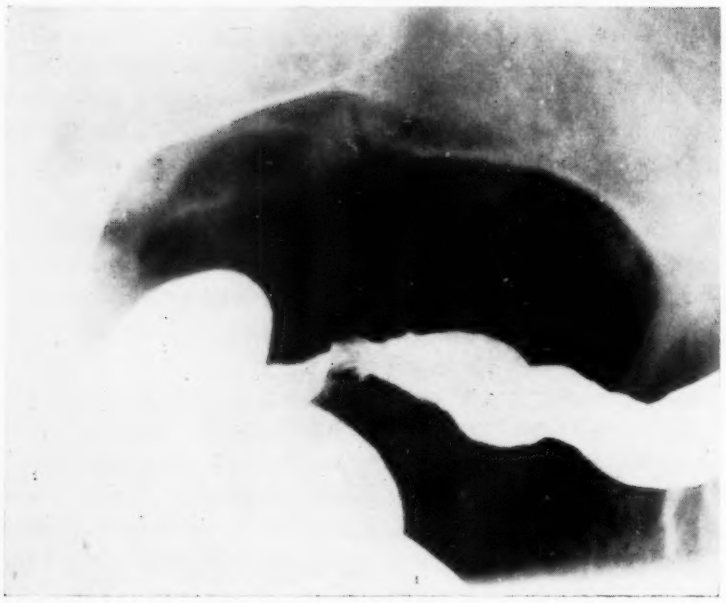

Fig. 1.—Roentgenologic appearance of a factitial (irradiation) stricture in the lower part of the sigmoid.

Fig. 2.—Section removed from the area of factitial (irradiation) proctitis. Note the thickening and hyalinization in the submucosa (×7).

Histopathologic Aspects of Specimens With Gross Ulceration.—Blocks of tissue were removed from the center of the base of the ulcers, from the margin of the ulcers, and at 1, 2, 3, and 5 cm. from the margin of the ulcers. As the distance from the ulcers increased, the histopathologic changes became less; often, at a distance of 3 to 5 cm., the tissues appeared to be normal.

Connective Tissue: Changes in the connective tissue were present in the mucosa, submucosa, stroma of the muscle, walls of vessels, and adventitial or serosal tissues. Some degree of edema usually was present, and the collagenous tissue was hyalinized. The collagen bundles were swollen, glossy, and afibrillar. This hyaline change suggested "setting" or "gelling" of the edematous connective tissue. These changes increased the thickness of the wall of the bowel, especially in the submucosal layer (Fig. 2). Actual fibrosis was absent except in the margin of the ulcers. Fibrosis should not be considered a factor in the production of the increased mass of hyaline connective tissue or increased thickness of the wall of the bowel. The processes of the fibroblasts were large and distorted. The nuclei were swollen, sometimes lobulated, and even multiple, with prominent dark nucleoli standing out against a pale background in "owl's eye" manner. No mitotic or proliferative activity was discovered.

Vessels: The telangiectatic blood vessels constituted a striking feature (Fig. 3, *a* and *b*). The walls of these vessels were thin, composed only of endothelial cells. In the vessels with walls composed of intimal, medial, and adventitial layers, there were varying degrees of hypertrophy of the intimal layer with resultant narrowing of the lumen. Some vessels were completely occluded by hyaline thrombi as a result of this intimal hypertrophy. These vascular changes were present in sections of nonulcerated tissue, as well as in the margin of the ulcers. As the distance from the ulcers was increased, the vascular changes grew less; at about 5 cm. they usually were absent.

Mucosa: In the ulcers the epithelial lining was destroyed. Evidence of regeneration and healing was minimal. Flat or cuboidal undifferentiated epithelium grew in for only a short distance from the edge of the ulcers. The glands in the mucosa at the margin of the ulcers were only slightly hyperplastic (Fig. 3, *c*).

When the mucosa was not ulcerated, it underwent marked alteration. Mucous stasis with overproduction was a common finding. The goblet cells were large and full, and were present in great numbers. The glands showed various forms and degrees of distortion. Some were short and wide-mouthed, some were narrow and elongated, while others were dilated and cystic. Among the cells atypical forms were seen frequently and even multinucleate and giant-cell elements were observed.

Muscle: The muscular tissue was altered in many instances. Edema and vacuolation of the fibers were present sometimes and, in instances of marked change, degeneration and atrophy of the fibers were prominent. Hyalinization of the interstitial connection tissue accompanied these changes.

Nervous Tissue: The intrinsic ganglionic nerve plexuses stained darkly and were prominent against the pale, edematous, hyalinized connective-tissue background, especially in the submucosa. In the marked reactions the ganglion cells appeared distorted, vacuolated, and degenerated.

Ulceration and Inflammation: Necrosis was present in the ulcers. The necrotic tissue usually presented features which were characteristic. There was a distinct, homogeneous, acidophilic, avascular structure with minimal inflam-

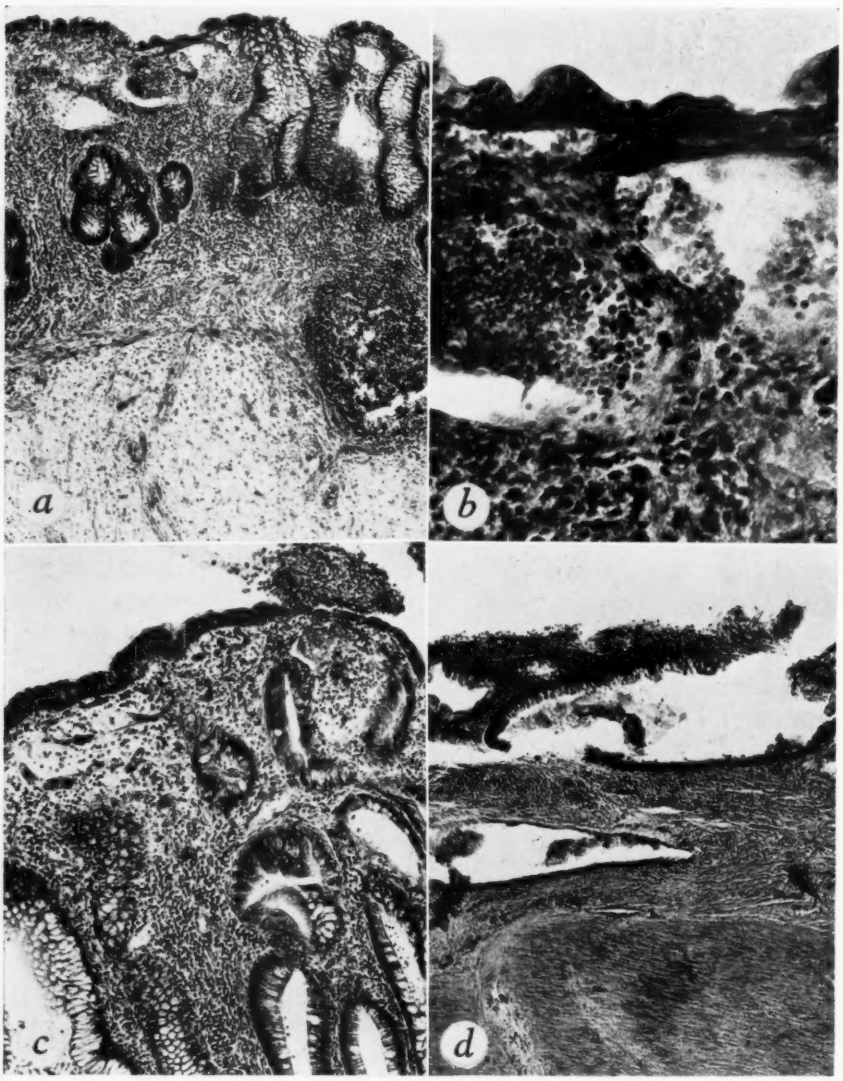

Fig. 3.—*a,* Section from an area of factitial (irradiation) sigmoiditis: note telangiectasis in the mucosa and derangement of the mucosal glands; a portion of the edematous hyalinized submucosa also is demonstrated (×50); *b,* a higher-power magnification of a portion of the mucosa shown in *a,* to demonstrate better the thin-walled telangiectatic vessels (×245); *c,* the mucosal edge of a factitial (irradiation) ulcer: note the limited amount of regeneration (×65); *d,* factitial (irradiation) proctitis, showing atrophic mucosa, telangiectatic blood spaces, and hyalinization of connective tissue and muscularis (×50).

matory cell infiltration. Sometimes there was no inflammatory cell infiltration. The cell outlines in these areas were still vaguely visible and appeared as "ghost" cells.

The inflammatory reaction was not marked and was seen only in the margin of the ulcers. The inflammatory cell infiltration extended from the edge of the ulcers only a very short distance under the intact mucosa. The plasma cell was the predominant cell type in these infiltrations. Only a minimal amount of granulation tissue was present.

The serosal tissue in the region of the ulcers was markedly thickened by fibrinous deposits and by hyaline changes in the connective tissue. The attached fat and mesenteric tissue in immediate relation to the wall of the bowel were liberally infiltrated with a mixed type of inflammatory cells and the trabeculae of connective-tissue stroma were thickened.

Histopathologic Aspects of the Specimen With Stricture Without Ulceration.—Blocks of tissue were removed from the area of greatest constriction, and at 2 and 4 cm. from this area. Marked alteration was present in all of the coats of the wall of the bowel except in the blocks of tissue removed at a point 4 cm. from the area of greatest constriction. No inflammatory reaction was seen in any of the sections.

There was marked atrophy of the mucosa (Fig. 3, *d*). In many small areas the epithelial cells lining the surface of the mucosa were flat to cuboidal in shape. No hyperplastic or regenerative changes were seen in the mucosal glands. Telangiectasis was present.

The submucosa was composed of dense, poorly cellular, hyalinized tissue, but it was not increased in thickness. No enlarged atypical fibroblasts, such as were seen in other specimens, were present. The most striking change was the presence of large, irregular vascular spaces with walls composed only of endothelial cells. All but two of the vessels with intimal, medial, and adventitial layers were occluded by hyaline thrombi which appeared to be the result of intimal hypertrophy. In several areas the submucosal tissue resembled amyloid deposit, but was negative when a stain for amyloid was used.

The muscular tissue and the intermuscular connective-tissue stroma were hyalinized. In many areas the muscular tissue was replaced by a homogeneous hyaline tissue. The tissue adjacent to the wall of the bowel was hyalinized, and the majority of the vessels in this tissue were completely occluded. Those which were not had decreased lumens brought about by hypertrophy of the intimal tissues.

Histopathology of the Specimens Removed for Biopsy.—These specimens had been removed from the margins of ulcers and fistulas during proctoscopic examination. They consisted of bits of mucosa and portions of the underlying tissue. Most of the specimens were taken six to nine months after completion of irradiation. The most prominent and constant feature was the telangiectatic vessels with walls composed only of endothelial cells. Hyperplasia of the epithelial cells and mucosal glands was present in many of the sections. In only one instance was a malignant process found. The specimen in this instance had

been removed from a rectovaginal fistula that appeared three months after irradiation of a cancer of the uterine cervix.

TREATMENT

Medical Treatment.—Treatment should be aimed at control of the distressing symptoms. Usually, patients can be taught proper care of the rectum, and can gain much in that manner. They must be informed that treatment generally is prolonged, occasionally for as long as a year or more, before relief can be expected. When acute, distressing symptoms are present, the discomfort as well as the mental anguish may be extreme. Patients should be reassured. They should be made to understand that the condition as a rule responds favorably to treatment.

Rest is essential. A diet that is adequate but low in residue should be prescribed. Often some form of sedative may be necessary. Belladonna and opium, administered either in suppositories or orally, may be required to control tenesmus and pain during the most acute phase of the condition. Intrarectal therapy has value. Warm (110° F. or 43.3° C.) olive oil containing a suspension of bismuth instilled into the rectum is comforting. Some believe that cod-liver oil is more efficacious. Warm mineral oil and warm witch hazel also are helpful. Two to 3 fluid ounces (60 to 90 c.c.) of any of these medicaments may be injected into the rectum through a soft rubber catheter. The patient should retain the liquid as long as possible. Patients who suffer from tenesmus may experience difficulty in retaining the liquid, but usually after a time they are able to retain it when given proper instruction and assistance.

From the beginning, warm, small, cleansing enemas of plain water at 110° F. should be used after each defecation to keep the rectum as free of fecal matter as possible. This should be done before each injection of medicated solutions. By following this plan, the patient will find it much easier to retain the liquid in the rectum. Ultimately, many patients can clear the bowel after defecation each morning, and the solution injected after irrigation can be retained in the rectum until defecation next morning. This is the objective of this phase of the treatment of factitial conditions, but the response of the acute rectal symptoms to this plan varies. Most patients note improvement after institution of the treatment, but often complete comfort may not come before the elapse of several months or longer. Apparently, the rapidity of symptomatic improvement is not always influenced by the type of factitial lesion present. The response of the acute symptoms when ulceration is present is about the same as it is when ulceration is absent. Likewise, improvement is not necessarily concomitant with healing of the lesion. Often patients are relieved of their subjective symptoms before complete healing of the lesion occurs. No type of cautery should be applied to a factitial lesion, and only the mildest solutions should be used in irrigations or retention enemas.

A most important phase of this treatment is constant supervision of the patient by the physician during the early period of management. The response to treatment varies widely, and if one method produces poor results, it is important to know it early. Difficulty may be experienced by some patients when

they are taking the small cleansing enemas preparatory to the injection of medicaments. Such an experience may defeat the entire program. Patients should be seen every day, and in some cases they should be confined to the hospital where they can be assured of proper care until they learn how to take care of themselves. The indiscriminate release of opium-laden suppositories to patients for the purpose of providing a method of relief from discomfort should be condemned.

Surgical Treatment.—Surgical treatment is employed only when complications arise, such as (1) obstruction, (2) hemorrhage, and (3) perforation and peritonitis. In such cases surgical treatment may be required as a life-saving measure. In a second group of cases surgical operation is employed in order to make it possible for the patient to live more comfortably. For example: (1) a stenosed segment of bowel may be removed; (2) sigmoidostomy may be performed to mitigate some of the unpleasantness caused by a rectovaginal fistula; (3) a rectovaginal fistula may be repaired; or (4) a stricture may be dilated or enlarged by a plastic type of surgical operation.

Before surgical closure of a rectovaginal fistula is attempted, enough time should be allowed to elapse to permit the diseased tissues to recover. During

TABLE III. FACTITIAL (IRRADIATION) PROCTITIS: SUMMARY OF 14 CASES IN WHICH OPERATIVE INTERVENTION WAS EMPLOYED

CASE	LESIONS	SURGICAL PROCEDURES	POSTOPERATIVE FOLLOW-UP		
			PRESENT CONDITION	TIME SINCE OPERATION	
				YEARS	MONTHS
1	Stricture of upper rectum; rectovaginal and vesicovaginal fistulas	Segmental resection of stricture with primary anastomosis; repair of fistulas	Well	10	1
2	Stricture of rectosigmoid	Segmental resection with primary anastomosis	Well	5	6
3	Stricture of rectosigmoid	Segmental resection with primary anastomosis	Well	3	4
4	Stricture of rectosigmoid	Extraperitoneal resection	Well	5	10
5	Ulceration with severe, repeated hemorrhages	Colostomy; later, combined abdominoperineal resection	Well	2	8
6	Stricture of upper rectum	Colostomy; strictureotomy; colonic stoma closed	Well		2
7	Stricture of upper rectum	Colostomy; closure of colonic stoma	Unknown		
8	Stricture of upper rectum	Colostomy*	Alive	13	
9	Ulcerated stricture of mid-rectum	Colostomy*; closure of colonic stoma	Rectovaginal fistula†	4	1
10	Stricture of sigmoid	Colostomy*	Well	5	
11	Rectovaginal fistula	Colostomy; repair of fistula; closure of colonic stoma	Well	3	1
12	Rectovaginal fistula	Colostomy	Fistula present	1	6
13	Rectovaginal fistula	Colostomy*	Fistula present	4	
14	Postradiation ileitis	Ileocolostomy	Well	1	8

*Operative treatment performed elsewhere.

†Fistula appeared eight months after closure of colonic stoma.

this period the fistula may decrease in size. When an optimal or elective surgical procedure is contemplated, the status of the original pelvic pathologic condition should be determined. If cancer persists, surgical procedures which are not required should not be employed. A summary of the surgical procedures employed in the treatment of the 14 patients in this group is given in Table III.

PROGNOSIS

If the malignant lesion has been destroyed, the factitial lesion usually will heal. Sometimes one or more years may elapse before healing is complete. A rectovaginal fistula or a stricture may develop and may persist. Some strictures may retrogress and some rectovaginal fistulas have been known to close spontaneously. Rectal bleeding arising from the telangiectatic vessels in the mucosa will persist long after a lesion has healed. This presents an unfortunate problem, and because of it, many patients become "rectum conscious" and are never relieved of concern.

COMMENT

The sensitivity of the intestinal mucosa to irradiation has been understood for a long time. Susceptibility in some individuals cannot be determined before irradiation, but this factor, too, has been suspected for a long time. Experimentally, these reactions have been demonstrated.

When a patient has symptoms related to the terminal portion of the colon a history of pelvic irradiation always should direct attention to the possibility of factitial disease.

The factitial lesions in our series were diagnosed by proctoscopy. Roentgenologic studies of the bowel are of value in certain cases in determination of the length of the involved segment, and in a few others in discovery of a lesion beyond the reach of the proctosigmoidoscope.

The character of a malignant lesion may be such that the adjacent normal tissues cannot be protected when therapeutic doses of radium are applied. No correlation of the dose of irradiation with the incidence of factitial lesions has been ascertained. The possible occurrence of factitial lesions should be understood when patients submit to treatment for malignant disease, but it should also be understood that such lesions are associated only with insignificant morbidity when they are compared to that of malignant processes.

The role of vascular changes in the production of the ulcerative lesions that appear months or years after the completion of irradiation has been the subject of much discussion. Some authors believe that vascular thrombosis caused by hyperplasia of the cells in the intimal layer in the small branches of the hemorrhoidal vessels results in an infarction of the involved tissue, and that this in turn produces ulceration. Others believe that vascular thrombosis plays an insignificant part in the pathogenesis of the ulceration. They admit that thrombosed vessels are present in the sections of tissue removed from the areas of injury, but they have found vessels with patent lumens as well. A study of the vascular changes in the sections of tissue removed from pathologic specimens with gross ulceration in this series showed that hypertrophy of the intimal tissue,

thrombosis, sclerosis, and degeneration become more marked as the margin of the ulcer is approached. However, a few vessels with decreased but patent lumens were seen in all of the sections in which thrombosis was a prominent feature. Thrombosis caused by marked intimal hypertrophy was very prominent in the sections taken from the bases and margins of the ulcers. Usually, at 3 to 5 cm. distance from the margins of the ulcers the vessels appeared normal. In the sections of tissue removed from the specimen with stricture without gross ulceration, there was complete occlusion of all but 2 of the vessels with medial and adventitial coats. These particular vessels had been present at the time of irradiation. The only other patent vascular channels seen were numerous large irregular telangiectatic spaces with walls composed only of endothelial cells. Therefore, it would seem that vascular occlusion induced by irradiation plays a significant role in the pathogenesis of ulcers and strictures which develop months and years after irradiation. If the ulceration does not develop as a result of an area of infarction in the mucosa, then trauma and infection superimposed upon an already damaged area of bowel in which the blood supply is limited are significant etiologic factors.

CONCLUSIONS

Sufficient irradiation to effect a cure of extrarectal pelvic cancer cannot be applied without the risk of damage to the intestines. The incidence of such injuries cannot be determined without routine diagnostic studies of the intestinal tracts of all patients in whom significant intestinal symptoms develop.

The appearance of intestinal symptoms at any time after pelvic irradiation suggests the possibility of factitial disease.

Factitial disease warrants a place in the differential diagnosis of lesions of the rectum and sigmoid. Careful studies serve to distinguish irradiation injuries of the rectum and sigmoid from extensions of pelvic malignant processes and from primary malignant lesions of these segments of the bowel.

Success in the treatment of irradiation injuries of the rectum and sigmoid depends upon recognition of the condition, well-planned treatment, and, occasionally, surgical operation.

Certain histologic characteristics, although not pathognomonic, are specific enough to be highly suggestive of irradiation effect on the tissues of the bowel. The primary changes to be looked for are (1) peculiar hyaline change in the connective tissue, (2) abnormal fibroblasts, (3) telangiectasis, (4) hypertrophy of the intimal layer of the blood vessels, (5) changes in the muscular tissue, and (6) epithelial and mucosal changes.

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Book Reviews

L'ostéosynthèse Au Clou. By R. Soeur, Chirurgien-Adjoint des Hôpitaux de Bruxelles. Pp. 130, with 134 illustrations. Paris, 1946, Masson & Cie, Editeurs Libraires de l'Academie de Médecine, Les Editions "Acta Medica Belgica", Bruxelles, Belgium.

This book is another contribution to the growing literature dealing with the intramedullary fixation of fractures of the long bones by means of intramedullary nails. The method, described by Küntscher in 1940, was introduced into France by surgeons in the German armies of occupation. The experience of the author with the method began in June, 1943. He has since described his results in fifty-five cases in the American literature.* This monograph presents essentially the same material greatly enriched by the addition of numerous illustrations. The apparatus used for the reduction, the positioning of the patient for operation, and roentgenograms of selected cases are carefully illustrated. The result is a graphic presentation of technical details of great value to any surgeon who wishes to essay the method. Appended is a short discussion of fractures of the neck and of the trochanter of the femur.

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*R. Soeur: Intramedullary Pinning of Diaphyseal Fractures, J. Bone & Joint Surg. 28: 309, 1946; Internat. Abstr. Surg. 83: 279, 1946.

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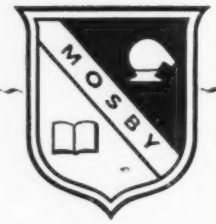

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